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Improved Communication

—HYGIENIC STANDARDS FOR DAILY INHALATION—

The Donald E. Cummings Memorial Lecture

HENRY FIELD SMYTH, JR., Ph.D.
Mellon Institute and Union Carbide and Carbon Corporation
Pittsburgh, Pennsylvania

EXPERIENCE convinces us that we humans are unique in the universe. Because of our ability to communicate, we may be well on the way toward emerging into a new level of biological existence. Each individual may eventually share completely all past and present experiences of the species. Each may act in concert with his fellows toward common ideals and goals, still retaining his own individuality. Real progress in this direction has been made during the past ten thousand years through developments of recording, duplicating and retrieval techniques. Despite brief back-sliding, there has furthermore been real spiritual progress, and an increase in the proportion of men of good will. The next ten thousand years should bring substantial achievements in communication upon higher levels, perhaps even through inarticulate contact of mind with mind. There are hints that what some have called the world mind may come into being before the present human species evolves physically into whatever new species its body is tending toward.

Henry F. Smyth, Jr.

However, until the world mind develops, we are forced to depend upon more prosaic means of communication. Not so many generations ago, a natural philosopher like Roger Bacon, whom today we call a scientist, could live a full life investigating the secrets of nature, feeling no need and finding no opportunity to communicate his discoveries and his conclusions to a living soul. He could bury his achievements in code, making them difficult for posterity to unravel. We do not have such people and such situations today. Each one of us benefits from the current division of labor, of experience, and of knowledge. Each one of us is a unique specialist, depending upon a multitude of other unique specialists for the achievement of our aims, for our very existence. If nothing else motivates us, simple self-interest should dictate that each one of us ought to make public all that he has learned, in order that his fellow specialists may use it to help us all.

Presented at the Seventeenth Annual Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, Philadelphia, April 25, 1956.

Communication

A YEAR AGO Sterner (1955) expressed the situation in more concrete terms. We comprise persons separately trained in highly specialized fields, led after training to cooperate in the common aim of providing means by which technological developments in occupation may be utilized in a manner compatible with complete health. He said, "We must provide a fluid and effective means of communication between the chemist, the engineer, the physicist, the toxicolo-

gist, the physician, and the other specialists brought into industrial hygiene. There must result from this interchange of ideas not only an appreciation of each team member's contribution, but an ability actually to bridge the gap between the disciplines, to synthesize, from the offerings of each of the fields, the solutions to the ever more complicated problems. Each member specialist must not only contribute the information he is most qualified to give, but also must encourage a sympathetic, intelligent, and mutual understanding."

In this matter of mutual understanding, we are very much like the inhabitants of Looking Glass Land. You remember that the White Queen told Alice, "Now *here*, you see, it takes all the running you can do, to keep in the same place. If you want to get somewhere else, you must run at least twice as fast as that." With the entry of new people into our profession and the recognition of new constituent specialties such as health physics and atmospheric pollution control, each with a tendency to keep to itself, are we running fast enough even to stay in the same place? We all feel there are too many conferences and too many committees to leave us time to do our daily work. On the other hand, if we are to do our work as well as it can be done, we must be in constant communication with related specialists, because each one of us fully knows only one facet of his own problems.

Communication is not a simple process. It requires an informed speaker or writer who can express himself at the level of comprehension of his audience. It requires an audience which wishes to receive communication. It must be carried on with words, those abstract symbols for reality, each of which has a different meaning to each individual, shaded by his entire past experience. Only a newly coined word is free from ambiguity, and it remains new for only a brief interval. Most important of all, successful communication requires what is today known as feed-back. By this, the speaker hears his audiences' impressions. He can correct and amplify his words until he thinks his audience truly perceives his meaning. A leisurely conversation can be effective communication through feed-back; an article in a journal is likely to be poor communication because feedback is inadequate.

The relatively new specialty of industrial toxicology has already contributed to the equally new profession of industrial hygiene by means of communication. The toxicologists are doing an acceptable job for those people who recognize their need for toxicological information and opinion. The job could be done better, but it is at least acceptable. Success is by no means as great in helping those people who do not recognize their need for help. Once more communication cannot succeed unless the audience desires to receive communication.

Acceptable Concentrations

THE MOST important communication within industrial hygiene, and between our profession and others, may be the collection of judgments upon acceptable concentrations of contaminants in working atmospheres. During what may be called the age of chaos, every experienced industrial hygienist had a few values uniquely his own, drawn from his own experience. For less familiar substances, he borrowed more or less judiciously from the values cherished by his professional colleagues. Some degree of unanimity was brought about when the United States Public Health Service values, based on its long-time collective experience in industrial hygiene, were published in a manual (USPHS 1943). Further unanimity followed publication of the values collected and extended by Cook (1945). In 1947 the American Conference of Governmental Industrial Hygienists published its first list in the Industrial Hygiene Newsletter (ACGIH 1947). In the next two years (ACGIH 1948, ACGIH 1949) revised lists were privately circulated to the members of the Association. Then (ACGIH 1950) publication took place in a scientific journal, and each year thereafter a revised list of threshold limit values has appeared in the scientific literature, and has been generally accepted.

The contributions of Cook (1945) in unifying opinion, and in weighing threshold limit values then in use, judging new data and proposing a list of 129 values, are worthy of high regard. Among the 238 values for substances other than mineral dusts in the current list (ACGIH 1956) of established and tentative threshold limit values are 54 of those which were first pro-

posed in Cook's list, some as definitely established, others to be used cautiously until verified by actual experience.

Not since Cook has anyone published a summary of the data which serve as bases for the selection of specific threshold limits. The privately circulated documents which give some of these data (ACGIH 1953b, 1954b, 1955b) cannot be considered to be publication, although they are freely available to any person.

Threshold limits are, and must continue to be the products of judgment, important if true. Some few truly represent their definition and are approximations of the maximum concentrations which can be inhaled continuously and repeatedly without injury to health. These may possibly be fit parameters for incorporation into codes and regulations. Many of the threshold limits are well below concentrations which can injure health. They represent current judgment as to concentrations to which, good practice dictates, men may be expected to subject themselves. These do not seem fit parameters for regulations. In a particular operation, if possible, it is desirable to maintain concentrations below the bench mark by reasonable ventilation and precaution. It is always best to reduce exposure to chemicals to the lowest practical level.

Previous Suggestions for Improvement

DURING the Ninth Annual Congress on Industrial Health, the writer was chairman of the Committee on Chemical Agents (1949). The report of the 16-man committee devoted considerable attention to praising the development of threshold limits, and to suggesting ways in which their presentation could be made more useful. Since that report, two developments have taken place along lines desired by the Committee. The annual table of the Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists is now published in the Archives of Industrial Health, removing the earlier implication of quasi-legal status arising from its appearance in the Industrial Hygiene Newsletter of the Division of Industrial Hygiene, U.S. Public Health Service. In 1954, the Committee on Threshold Limits of that Association began to supplement its table of accepted values with a list of tentative values.

Three other suggestions of the Committee on Chemical Agents deserve reiteration and discussion. It was urged that the bases for the selection of each value should be published, that the name should be changed to *hygienic standard*, and that the particular concept of permissible human response behind each value should be clearly indicated.

There is such a multitude of factors involved in the protection of health in our complex civilization that no one person or group of persons is competent to weigh them all with assurance. No oracular or *ex cathedra* statement on health deserves serious attention. Only when the facts upon which a decision are based are furnished for general scrutiny and evaluation can the decision be considered even tentatively sound, and only after there has been adequate opportunity for criticism and modification can it be considered established. All toxicological facts should be published, and all decisions upon the facts should be accompanied by a summary of the reasoning under which they were derived, before anyone should be expected to act upon the decisions. Any publication of standards for maintenance of health ought to include reference to the underlying data.

The second suggestion referred to the name by which the values are known. Semantics is more than a sport for the idle. No matter how thoroughly a concept is originally presented, it always becomes known and referred to by a brief name, a catchword. Most persons who learn of the concept hear the catch-word name, and do not go back to the original presentation. The meaning they attach to the name comes from their previous experience with the particular words. It may be, but is usually not, exactly what the originator of the idea intended. The more carefully one chooses the name he assigns to a concept, the more likely are others to interpret the concept as he himself does.

The values now known as threshold limits are usually identified by phrases containing the words *allowable* or *permissible*. These two words have connotations of legal regulations. Such connotations cannot properly attach to the judgment of a voluntary professional association. The identifying phrases may also contain the words *maximum*, *threshold* and *limit*. These words all

imply that below the concentration specified, human response is negligible, above the concentration it is dangerous. Actually, it is more than an implication. It is definitely stated. In the introduction to its 1956 list (ACGIH 1956) the Committee on Threshold Limits says, "Values are given . . . for the maximum average atmospheric concentrations of contaminants to which workers may be exposed for an eight-hour working day without injury to health." Careful study of the data which support the currently accepted values suggests that no such description can be truthfully attached to most of them. Industrial hygienists recognize this. They are accustomed to emphasize that the values should be regarded simply as bench marks, guides to good practice. Indeed, the Threshold Limits Committee itself confusingly warns "Threshold limits . . . should not be regarded as fine lines between safe and dangerous concentrations." (ACGIH 1956)

The term *maximum acceptable concentration* being used in revisions of standards by the American Standards Association Z-37 Committee is objectionable only because it will be abbreviated M.A.C. Many will interpret this abbreviation as *maximum allowable concentration,* and nothing will have been gained by the change from *allowable* to *acceptable.*

I conclude that the names *maximum allowable concentration* and *threshold limit* are misleading. They convey a wrong impression to those who are not already familiar with the concepts behind the values. The name suggested in 1949, *hygienic standard,* is not misleading. Standards of good practice are familiar to all of us in many fields. Looking toward the future provision of a variety of hygienic standards, a series of values should be selected, to be known as *hygienic standards for daily inhalation.*

The third suggestion is more far-reaching. The Committee on Chemical Agents (1949) pointed out that there has been no simple or uniform relation between the effects of a substance and the numerical value chosen for tabulation. The Committee concluded that concentrations have been selected on the basis of one of four concepts of the level best suited to hygienic control of inhalation, the choice having been governed by the nature of the toxic response and by the degree of organoleptic response. The Committee's four concepts follow:

a. *Plus or minus*: The maximal time-weighted average concentration which produces only minor injury, and that in a very small proportion of exposed workmen.

b. *Safe*: The maximal time-weighted average concentration which sound evidence leads one to believe will cause no demonstrable illness or other symptom of toxic effect in any workman during a lifetime of industrial exposure.

c. *Bench mark*: A concentration based on the belief that any unnecessary exposure is undesirable—a concentration lower than that of *a* or *b*, one as low as is consistent with practical engineering control.

d. *Comfort*: A concentration lower than *a* or *b*, representing the maximum which in a short time is not objectionable to 9 out of 10 of a group of persons not accustomed to inhalation of the substance.

Note well that these four concepts were judged to be those already used for the selection of hygienic standards for daily inhalation. All four were judged consistent with the goals of industrial hygiene.

Hygienic Standards for Daily Inhalation

THE SUBJECT of hygienic standards for daily inhalation should be re-examined, the concepts represented by the values should be restated in more realistic toxicological terms, and more consistent and more informative standards should be prepared. Such a step will not undo any of the accomplishments of the profession of industrial hygiene or of any organization. Rather, it will supply informative standards to supplement the accumulation of naked numbers now accepted, some of which have not been critically re-examined for a decade.

It is certainly imperative that the inhalation of substances during the working day shall not be allowed to result in any injury to the physical well-being of workmen. It is furthermore imperative that inhalation shall not increase the probability of accidents through the mental distress occasioned by objectionable eye, nose or throat irritation, transient though it may often be, nor through the impaired judgment and delayed reaction time of light narcosis. It is desirable that inhalation shall result in no degree of discomfort whatsoever. On the

other hand, when it is impractical to avoid all discomfort, then such inhalation is certainly justified, provided there results no injury to workmen and no increase in the probability of accidents.

Tables of hygienic standards do not now carry indications of the nature and of the magnitude of the effects to be expected from inhalation of greater concentrations. It is only by a rather thorough study of the available data that one can decide whether or not a particular substance can safely be inhaled at a greater concentration. With most substances, it is quite practical to set two standards, one an inoffensive level, another a concentration which cannot safely be exceeded under any pressure of practicality.

Administrative expediency may be served by a table of numbers which constitute a part of official regulations. Regulations need not be defended, they need not cite justifications. However, it is a minority of the profession of industrial hygiene who have regulatory responsibilities. Most of our colleagues act through obtaining voluntary cooperation with their judgments. They would be aided by a greater degree of explanation in a tabulation of standards. They could then show that their recommendations are quite defensible, that they are not arbitrary decisions having no regard for the realities of competitive industrial existence.

A hygienic standard for daily inhalation should specify two concentrations, together with a description of the human response to be expected from inhalation of each. One concentration should be low enough so that no injurious effect can be expected in any workman, but it may have a detectable odor, it may cause a detectable eye, nose or throat irritation. The second concentration should produce somewhat more severe, but still reversible and non-progressive effects. From these two concentrations, one can at once see how strictly the standard should be observed, the steepness of the dosage-response curve, the breadth of the plateau of non-injurious concentrations. Despite the wide range in individual susceptibilities of workmen, such values can be selected for most, if not all, substances.

Adequate data for preparing this sort of standard consist of appropriate human experience, or repeated medical examinations of workmen in atmospheres whose concentration has been frequently estimated. Sterner (1955) discussed this point in detail. Experiments upon animals, verified by biochemical and physiological studies in humans, may demonstrate conclusively that a substance has an effect during one period of inhalation which does not progress during oft-repeated inhalation. For such a substance, satisfactory data can be obtained from objective study and secretly recorded subjective effects of a group of humans inhaling known concentrations for substantially eight hours. Any lesser body of data should result in a hygienic standard being designated as tentative.

Categories of Objectionable Action

JUDGMENTS should be made to determine which hygienic standards for daily inhalation must be carefully observed, and which may be exceeded when it is impractical to observe them. These judgments will be most consistent if we first decide for each substance what objectionable action we are guarding against by the standard. Every toxicologist will realize that the action at a low concentration which it is most important to guard against, may not be the same as the menace to life to be expected at a high concentration. In a tentative fashion, the writer has made these decisions for the 238 substances, exclusive of the mineral dusts, included in the 1956 tables of proposed accepted and tentative standards (ACGIH 1956). The decisions can be divided into the following nine categories on the basis of the nature of human response.

Chronic toxicity. The most dangerous effect of some substances is a progressive systemic injury, increasing in severity with continuing inhalation. Benzene, carbon disulfide, carbon tetrachloride and lead are the most familiar examples. The lower standard for these substances should be a concentration believed not to produce any effect in any workman, and no considerations of practicality are sufficient to justify inhalation in excess of the standard. Close medical supervision is required for safe use of these substances. The standard for chronically toxic substances should refer to the time-weighted average concentration throughout a working day. Brief peaks of a few times the standard have no significance, save as they increase the average.

Acute toxicity. Some substances do not produce an injury progressing with repeated inhalation. Such systemic injury as they may cause takes place as the result of one excessive inhalation, or not at all. Familiar examples are carbon monoxide and hydrogen cyanide. The standards for acutely toxic substances should be interpreted in the same light as those for chronically toxic substances.

Narcosis. The most dangerous effect of some substances is narcosis, which becomes anesthesia in its extreme stage. At a rather low concentration they induce accidents by impairing judgment and delaying reaction time. Familiar examples are ethyl alcohol, ethyl ether and gasoline. The lower standard for a narcotic substance should be a concentration which produces no detectable effect upon judgment and reaction time after eight hours inhalation. It should refer to the average concentration existing during some appreciable period of time, the length of which can be estimated from absorption and elimination data. No considerations of practicality can justify exceeding the standard for a narcotic substance.

Irritation. The most dangerous effect of some substances is irritation. Eye, nose and throat are irritated at a low concentration, the bronchi at a higher concentration, and fatal lung edema may be the result of inhaling an extreme concentration. The aldehydes, halogens and acids are familiar examples. Highly odorous substances may also be considered in this category. The lower standard for an irritant substance should be a concentration which is detectable, but is not objectionably irritating to the majority of unhardened subjects who are exposed for a substantial part of a working day. The higher standard should be set at a concentration which is well under one injuring bronchi or lungs, and which is justifiable when it is impractical to keep concentrations at the lower standard. Standards for irritating substances should refer to concentrations existing for even a brief period during a work day.

Asphyxiation. Some substances are inert in the body and can injure only by asphyxia at extremely high concentrations, excluding the oxygen of the atmosphere. Familiar examples are the fluorochloro refrigerants. The lower standard for these asphyxiants should be a nominal bench mark of good engineering practice, such as the 1000 ppm concentration now quoted. The inert nuisance dusts like iron oxide might well be placed in this same category, and the currently used bench mark of 15 mg./cu.m. seems an appropriate level. The standard should refer to the concentration existing during any brief period, but it should be recognized that higher concentrations are justified when it is impractical to keep below the standard.

Fume fever. The most important effect of some substances is a transient influenza-like condition known as fume fever. A familiar example is zinc oxide fume. The lower standard for a fume fever producer should be a concentration which will not produce that distressing but not menacing condition in any workman, and it should apply to an appreciable period, such as half an hour. No considerations of practicality can justify exceeding the standard for fume fever producing substances.

Eye pigmentation. The most important effect of two substances, quinone and hydroquinone, appears to be a slowly developing pigmentation of the sclera, which may reduce visual acuity, or even lead to blindness. The lower standard for these substances should be a concentration which produces no pigmentation after years of exposure, and it should refer to the time-weighted average concentration throughout the day. For a few days at a time, conditions of practicality should justify exceeding the standard.

Cancer. One substance is reasonably well established as a cause of respiratory tract cancer. This is nickel carbonyl. It appears probable that the minimum cancerigenic exposure will never be defined. At this time it is prudent to set the standard for a cancerigenic substance substantially at zero, as has already been done for nickel carbonyl, and no considerations can justify allowing the inhalation of any concentration which is avoidable.

Allergy. Some substances are known to sensitize an appreciable proportion of exposed workmen. They may produce distressing and menacing asthma-like attacks when a sensitized person inhales a low concentration. Examples are ethylene diamine and the diisocyanates. At this time there is no

rational experimental basis for defining a concentration which will not sensitize a susceptible workman, or one to which no previously sensitized workman will respond. Control of exposure to allergenic substances must rely heavily upon industrial medicine. After experience has allowed withdrawal of workmen susceptible to sensitization, the remaining resistant individuals can be protected by a hygienic standard for daily inhalation based upon irritation or systemic injury. Until it has been demonstrated that a particular group includes no susceptible workmen, no considerations can justify allowing inhalation of any concentration which is avoidable.

Interpretations of Accepted Values

NO NEW VALUES for standards are suggested at this time. The available data upon which the 238 values in the 1956 proposed list (ACGIH 1956) appear to be based have been studied. Table I is offered as an interpretation of these values, increasing the information they convey. It presents the familiar numbers, which give the engineer and the chemist an illusion of complete understanding. It also presents, in the form of abbreviations of self-evident meaning, some description of actions which gives the biologically and medically trained a feeling of confidence. The table is obviously too complex for great popularity. Nevertheless, every class of information listed is required by those who must apply the values. The data relied upon for the interpretations and some comments on their adequacy are summarized after the table.

All substances in the proposed 1956 threshold limits table (ACGIH 1956), except mineral dusts, appear in one alphabetical order. When a value is listed in units of milligrams per cubic meter, the letter *m* precedes the number. When a tentative value was proposed the letter *T* follows the number.

Following the threshold limit values is a column showing a personal judgment of the most serious effect of inhalation of a concentration somewhat higher than the threshold limit. These judgments allow one to decide whether the value should refer to the time-weighted average concentration or to peak concentrations, existing at any time during the day. In future tables of standards the decision should be clearly indicated in the table.

Three columns record personal judgments and estimates as to what responses may occur in some workman inhaling continuously, all day, the threshold limit, twice, and ten times the limit. It will be obvious from these entries that the values may not always define concentrations in which workmen will find no objectionable sensory effect, or even concentrations where no toxic symptoms will develop in any individual.

Next comes a column listing important injuries other than from inhalation of the substance itself, such as dangerous absorption through the skin, chemical burns of eye and skin, frequent allergic dermatitis, pyrolysis to phosgene, and the like. The almost universal defatting of the skin by solvents, and freezing of tissue by low boiling liquids, has not been entered.

The last two columns give some indication of the soundness of the value by describing the supporting data, and by specifying the year in which it was first proposed or adopted. It is, of course, true that a value proposed many years ago and recopied in each succeeding year's list is not necessarily proven sound, but in general it is likely to be better established than a more recently adopted value.

There may be objection that the table does not mention warning power nor attempt to evaluate this property specifically. The practical importance of warning power in preventing inhalation of an excess is much over-rated. Odor data are notoriously unreliable. Estimates of tolerable working conditions with unacclimated subjects, briefly exposed, have only limited usefulness in predicting the responses of acclimated and usually hardened workmen, exposed all day. Early stages of narcosis reduce perception of odor and irritation. Even with strong irritants like ammonia and acrolein, physical circumstances, or a sense of duty, may keep a man at his post to be seriously injured by a concentration which, all would predict, cannot be inhaled voluntarily.

There is nothing in Table I which is not easily accessible, if not already well known to a thousand experienced industrial hygienists and toxicologists. Not one of these but will object to some among the thousand personal judgments entered. However, the

annual threshold limit tables are consulted by upwards of twenty thousand other persons who do not have access to original sources and extensive experience. These people rightly regard the threshold limits as a presentation of the best available judgment, and they may wrongly regard them as everything they need to know about safe use of a substance. Despite specific disclaimers, printed with each year's table, these readers tend to regard each threshold limit as defining the line between safety and injury.

Table I is presented as a beginning in the extensive job of improving communication by developing a rational and informative series of hygienic standards for daily inhalation.

Summary of Underlying Data

THE DATA relied upon for the interpretations entered in Table I are summarized and briefly commented upon in the pages which follow. It is believed that the most significant published information is included, but no attempt has been made to list all pertinent articles. Certain hitherto unpublished observations are included when they appear to confirm or supplement material in the literature. The term "most important effect" used with every substance, refers to the possible effect of inhalation of concentrations a few times the threshold limit, not necessarily to the possible effects at very high concentrations.

Acetaldehyde. Cook (1945) quotes the 1911 report of Iwanoff that cats inhaling 280 ppm for seven hours were not noticeably affected. The unacclimated subjects of Silverman, Schulte and First (1946) found 25 ppm objectionable, 50 ppm irritating to the eyes, but even 200 ppm not irritating to the throat. Fairhall (1949, p. 199) describes the effects as irritation, narcosis, bronchitis, albuminuria, fatty liver and lung edema. He concludes that inhalation does not cause chronic poisoning, and that death is due to anesthesia when prompt, or to lung edema when delayed. Smyth (1937-55) found rats survive four hours inhalation of 8000 ppm but die from 16,000 ppm. The liquid causes severe corneal injury, irritates the skin and may sensitize some persons.

MNEMONIC ABBREVIATIONS USED IN TABLE I.

acne—	chloracne from continued skin contact.
acute—	acute toxicity, with little or no increase in severity from continued inhalation.
all—	allergenic. Dermatitis and asthma-like sensitization may result.
ALL—	allergenic. Dermatitis and asthma-like[!] sensitization are likely.
asphyxia—	asphyxiation at very high concentrations.
bur—	burn of the skin.
BUR—	very severe burn of skin.
cancer—	cancer reported in humans.
chronic—	chronic toxicity, with increase in severity from continued inhalation.
clin—	clinical examination of workmen was correlated with their exposure.
cns—	central nervous system stimulation, such as tremors or convulsions.
cy—	cyanosis (blue skin) may be evident.
CY—	cyanosis (blue skin) may be marked.
est—	estimate from experience and analogy.
eye—	eye irritation severe enough to require medical treatment.
EYE—	eye burn may be severe.
eye pig—	eye pigmentation without injury.
fume—	fume fever.
head—	headache. This symptom has not been entered every time it may occur.
hu—	human sensory data.
HU—	human toxicological or physiological data.
ind—	industrial complaints or observations, less quantitative than clinical examinations.
irr—	irritation of eye, nose or throat in some.
IRR—	irritation of eye, nose or throat marked.
lung?—	minor irritation of bronchi (coughing) or lungs.
lung—	definite irritation of bronchi or lungs with injury of lungs possible.
LUNG—	dangerous injury of lungs with little warning.
m—	the quantity is expressed in milligrams per cubic meter (mg./cu.m.), not in ppm.
med—	medical uses yield some information.
narcosis—	narcosis, ranging from impaired coordination through dizziness, to anesthesia.
nar?—	faint narcosis, somewhat impaired reaction time and judgment.
nar—	narcosis definite, short of dizziness.
NAR—	narcosis marked, dizziness to unconsciousness.
nau—	nausea. This symptom has not been entered every time it may occur.
none—	no effects are expected.
odor—	odor may be perceptible.
ODOR—	odor marked.
pyr—	pyrolysis to lung injuring halogen compounds in a flame, or on hot metal.
rad—	radiation injury is possible.
rpt—	repeated animal inhalation results.
sgl—	single animal inhalation results.
skp—	skin penetration may cause symptoms.
SKP—	skin penetration of liquid is dangerous.
T—	tentatively proposed.
tox?—	toxic symptoms may arise very slowly.
tox—	minor toxic symptoms.
TOX—	major toxic symptoms.
vis—	visual acuity loss

TABLE I.
INTERPRETATION OF THRESHOLD LIMIT VALUES PROPOSED FOR 1956
Exclusive of Mineral Dusts
(Mnemonic abbreviations explained at foot of table)

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Acetaldehyde	200.	lung	IRR-odor		lung	all-EYE	hu-sgl	1945
Acetic acid	10.	lung	irr-odor		IRR-lung	bur-EYE	hu-ind-sgl	1945
Acetic anhydride	5.	lung	irr-odor		eye-IRR-lung	bur-EYE	sgl	1947
Acetone	1000.	narcosis	irr-nar-odor		NAR		HU-ind	1953
Acrolein	0.5	LUNG	irr	IRR	lung	bur-EYE	hu-sgl	1945
Acrylonitrile	20.	acute	none	odor	tox	skp	rpt	1943
Aldrin	m-0.25	chronic	none	none	irr-tox	all-skp	clin	1954
Allyl alcohol	5.	EYE-lung	irr		eye-lung	bur-EYE	ind-sgl	1954
Allyl chloride	5.	T lung	none		irr-odor	pyr	sgl	1956
Allyl propyl disulfide	2.	lung	irr	IRR			ind	1954
Ammate	m-15.	lung	none		lung		est	1954
Ammonia	100.	lung	irr-odor		IRR-lung	bur-eye	HU-ind-sgl	1943
Amyl acetate	200.	narcosis	irr-odor	IRR-nar	NAR		hu-sgl	1945
Amyl alcohol	100.	narcosis	irr-odor	IRR-nar	NAR		hu-sgl	1945
Aniline	5.	chronic	none	cy	CY-tox	SKP	rpt	1943
Antimony	m-0.5	chronic	none	none	tox		clin-rpt	1948
ANTU	m-0.3	T chronic	none		tox		est	1956
Arsenic	m-0.5	chronic	none	tox?	tox		clin-est	1948
Arsine	0.05	acute-lung	none		lung ?-tox		ind-sgl	1947
Barium (soluble)	m-0.5	acute-lung	none		tox		est	1943
Benzene	35.	chronic	tox?	tox	nar-odor-TOX		clin-rpt	1948
Benzyl chloride	1.	lung	irr	odor	IRR-lung	EYE	sgl	1954
Bromine	1.	lung	irr-odor		IRR-lung	BUR-EYE	ind-sgl	1945
Butadiene	1000.	narcosis	none	odor	nar		hu-rpt	1947
Butanone (methyl ethyl ketone)	250.	narcosis	irr-odor	IRR-nau	nar		hu-sgl	1948
Butyl acetate	200.	narcosis	irr-odor	IRR-nar	NAR		hu-sgl	1945
Butyl alcohol	100.	narcosis	odor	irr	IRR-NAR		clin	1950
Butyl amine	5.	lung	odor	irr	eye	BUR-EYE	ind-sgl	1955
Butyl CELLOSOLVE	200.	chronic	odor-tox ?	nar-tox	irr-nar-TOX	skp	rpt	1945
Butyl mercaptan	10.	T lung	ODOR		eye-irr-lung ?		sgl	1954
Cadmium oxide fume	m-0.1	acute-lung					ind-rpt	1943
Calcium arsenate	m-0.1	T chronic	none	none	none		est	1956
Carbon dioxide	5000.	asphyxia	none		nar		HU	1943
Carbon disulfide	20.	chronic	none	odor	nar-tox		clin-rpt	1943
Carbon monoxide	100.	acute	none	tox	TOX		clin-HU	1943
Carbon tetrachloride	25.	chronic	tox?	tox	nar-odor-TOX	pyr	clin-ind-rpt	1953
CELLOSOLVE	200.	chronic	odor	nar-tox	irr-nar-tox		rpt	1945
CELLOSOLVE acetate	100.	chronic	odor	irr-tox	IRR-nar-tox		rpt	1945

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Chlordane	m-2.0	chronic	none	none	irr-tox	all-skp	clin	1954
Chlorinated camphene (60%)	m-0.5 T	chronic	none		tox	all-skp	est	1956
Chlorinated diphenyl oxide	m-0.5	chronic	none	tox?	tox	acne	rpt	1955
Chlorine	1.	lung	odor	irr	IRR-lung	bur-EYE	hu-rpt	1948
Chlorine trifluoride	0.1	lung	none		irr-lung	bur-EYE	rpt	1955
Chlorobenzene	75.	narcosis	odor	irr-tox	nar	pyr	est-sgl	1943
Chlorobromomethane	400. T	narcosis	none	nar	tox	pyr	rpt	1956
Chlorodiphenyl (42% Cl)	m-1.	chronic	none	none	tox?	acne	rpt	1945
Chlorodiphenyl (54% Cl)	m-0.5 T	chronic	none	tox?	tox	acne	rpt	1956
Chloroform	100.	chronic	odor-tox?	nar-tox	TOX	pyr	est-med-sgl	1945
1-Chloro-1-nitropropane	20.	lung	none		irr-lung-tox		sgl	1945
Chloropicrin	1. T	lung	odor	irr	lung		clin	1956
Chloroprene	25.	chronic	none	nar-tox	TOX	pyr-skp	rpt	1945
Chromic acid, chromates as CrO_3	m-0.1	irr	none	irr	IRR	bur-EYE	clin	1943
CRAG herbicide	m-15.	chronic	none	none	irr-tox?		est	1954
Cresol	5.	chronic	odor	irr	lung-tox	BUR-EYE-SKP	est-sgl	1952
Cyanide as CN	m-5.	acute	none		tox		est	1947
Cyclohexane	400.	narcosis	none	nar-odor	irr-NAR-tox		rpt	1945
Cyclohexanol	100.	narcosis	irr-odor	nar-tox	IRR-NAR		hu-rpt	1945
Cyclohexanone	100.	narcosis	irr-odor		IRR-nar		hu-rpt	1945
Cyclohexene	400.	narcosis	none	nar-odor	irr-NAR-tox		sgl	1945
Cyclopropane	400.	narcosis	none	nar-odor	irr-NAR		est-med	1947
2,4-D	m-10.	chronic	none	none	irr-tox?		est	1954
DDT	m-1. T	chronic	none	none	irr-tox	skp	est	1954
Decaborane	0.05 T	acute	none		odor		rpt	1956
Diacetone alcohol	50.	narcosis	irr	odor	IRR-nar		hu-sgl	1954
Diborane	0.1	cns-lung	none		lung-tox?		clin-rpt	1955
o-Dichlorobenzene	50.	chronic	odor-tox?	irr-tox	nar	pyr	ind-sgl	1947
Dichlorodifluoromethane	1000.	asphyxia	none	none	none	pyr	rpt	1947
1,1-Dichloroethane	100.	chronic	odor		nar-tox	pyr	rpt	1945
1,2-Dichloroethylene	200.	narcosis	none	odor	irr-nar	pyr	sgl	1945
Dichloroethyl ether	15.	lung	none	irr-odor	lung	skp	hu-sgl	1945
Dichloromonofluoromethane	1000.	asphyxia	none	none	none	pyr	sgl	1947
1,1-Dichloro-1-nitroethane	10.	lung	none	irr	lung-tox		rpt	1945

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Dichlorotetrafluoromethane	1000.	chronic	none	none	none	pyr	sgl	1947
Dieldrin	m-0.25	asphyxia	none	none	irr	all-skp	clin	1954
Diethylamine	25.	lung	odor	eye-irr	IRR-lung	bur-EYE	sgl	1952
Difluorodibromomethane	100.	chronic	none	none	irr-nar-tox	pyr	rpt	1955
Diisobutyl ketone	50.	narcosis	irr-odor		IRR-nar		hu-rpt	1954
Diisocyano toluene	0.1 T	ALL-lung	all		ALL		ind	1956
Dimethylaniline	5.	chronic	none	cy	CY-tox	SKP	sgl	1943
Dimethylsulfate	1.	acute-LUNG	none		LUNG-TOX	BUR-EYE-SKP	sgl	1945
Dinitrobenzene	m-1. T	chronic	none		tox	skp	est	1956
Dinitro-o-cresol	m-0.2	acute	none		irr-tox	skp	sgl	1949
Dinitrotoluene	m-1.5	chronic	tox?	tox?	irr-tox	skp	est	1943
Dioxane	100.	chronic	none	nar-odor	irr-NAR-tox		hu-rpt	1947
EPN	m-0.5	acute	none		tox		est	1954
Ethyl acetate	400.	narcosis	irr-odor	nar	IRR-NAR		hu-sgl	1945
Ethyl acrylate	25. T	lung	odor	irr	lung		rpt	1956
Ethyl alcohol	1000.	narcosis	irr-odor	nar	IRR-NAR		HU	1945
Ethylamine	25.	lung	odor	eye-irr	IRR-lung	bur-EYE	sgl	1952
Ethyl benzene	200.	narcosis	nar?-odor	irr	NAR		hu-sgl	1945
Ethyl bromide	200.	lung	none	irr-odor	lung	pyr	sgl	1947
Ethyl chloride	1000.	narcosis	odor		nar	pyr	sgl	1947
Ethylene chlorhydrin	5.	acute	none	tox?	odor-tox	SKP	ind-sgl	1947
Ethylene diamine	10.	ALL-lung	odor	irr	eye	ALL-bur-EYE	ind-rpt	1955
Ethylene dibromide	25.	chronic-lung	none	irr-odor	lung-tox	pyr	rpt	1953
Ethylene dichloride	100.	chronic	odor-tox?	nar-nau	TOX	pyr	ind-rpt	1953
Ethylene imine	5.	acute-lung	odor		lung-TOX	bur-eye-SKP	hu-sgl	1955
Ethylene oxide	100.	lung-narcosis	odor-nar	irr	IRR-LUNG-NAR	bur	hu-rpt	1945
Ethyl ether	400.	narcosis	irr-odor	IRR	NAR		HU-med	1947
Ethyl formate	100.	narcosis	none	irr-odor	nar		sgl	1947
Ethyl mercaptan	250. T	lung	ODOR		eye-irr-lung		sgl	1954
Ethyl silicate	100.	acute-lung	odor	irr-tox	IRR		hu-rpt	1945
Ferbam	m-15. T	lung	none	irr	IRR		est	1956
Ferro vanadium dust	m-1.	lung	none		none	all	rpt	1954
Fluoride dust	m-2.5	chronic	tox?		irr-tox		clin-HU	1947
Fluorine	0.1	lung	none		irr-lung?-tox?	BUR-EYE	clin-rpt	1953
Fluoroacetates	m-0.1 T	acute	none		tox		est	1956
Fluorotrichloromethane	1000.	asphyxia	none	none	none	pyr	sgl	1945
Formaldehyde	5.	lung	irr	odor	IRR-lung	all-EYE	ind-sgl	1948
Furfural	5. T	lung	irr		IRR-lung	all-EYE	ind-sgl	1954
Furfuryl alcohol	50. T	narcosis	odor	irr-nar	IRR-NAR		rpt	1956
Gasoline	500.	narcosis	odor	irr-nar	NAR		hu-ind	1945

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Heptane	500.	narcosis	odor	irr-nar	NAR		hu	1945
HETP	m-0.1 T	acute	none		tox	SKP	est	1956
Hexane	500.	narcosis	odor	irr	irr-nar-nau		hu	1947
Hexanone (methyl butyl ketone)	100.	narcosis	odor	irr	nar		hu-sgl	1947
Hexone (methyl isobutyl ketone)	100.	narcosis	odor	irr	nar		hu-sgl	1947
Hydrazine	1.	lung	none		cns-irr-lung	bur-eye	rpt	1955
Hydrogen bromide	5.	lung	irr	IRR	eye-lung	bur-eye	hu	1955
Hydrogen chloride	5.	lung	irr	IRR	eye-lung	bur-eye	ind-rpt	1948
Hydrogen cyanide	10.	acute	odor		TOX	SKP	hu-sgl	1948
Hydrogen fluoride	3.	chronic-lung	irr	IRR	eye-lung-tox	BUR-EYE	ind-rpt	1943
Hydrogen peroxide, 90%	1.	lung	none		irr-lung	BUR-EYE	rpt	1955
Hydrogen selenide	0.05	chronic-lung	none		lung-odor-tox		hu-ind-rpt	1948
Hydrogen sulfide	20.	lung	irr-odor		lung-tox		clin-sgl	1943
Hydroquinone	m-2.	eye pig	none	eye pig	vis		clin	1955
Iodine	0.1	lung	irr	IRR	eye-lung	bur-eye	ind	1948
Iron oxide fume	m-15.	fume-lung	none	fume-irr	lung		clin	1947
Isophorone	25.	chronic-narcosis	irr-odor		IRR-nar-tox		hu-rpt	1945
Isopropylamine	5.	lung	odor	irr	eye	BUR-EYE	ind-sgl	1955
Lead	m-0.15	chronic	tox?	tox	TOX		clin	1943
Lead arsenate	m-0.15 T	chronic	none	none	irr		rpt	1956
Lindane	m-0.5	chronic	none	none	irr	all-skp	rpt	1954
Magnesium oxide fume	m-15.	fume	none	fume-irr	lung		HU	1945
Malathon	m-15.	acute	none		tox		est-clin	1954
Manganese	m-6.	chronic	none	none	tox		clin	1945
Mercury	m-0.1	chronic	none	tox	TOX		clin-rpt	1943
Mercury, organic	m-0.01	chronic	tox?		tox	SKP	clin-sgl	1954
Mesityl oxide	50.	narcosis	irr-odor	nar	IRR-lung		hu-rpt	1945
Methoxychlor	m-15.	chronic	none	irr-tox			sgl	1954
Methyl acetate	200.	narcosis	odor	irr	nar		est-sgl	1948
Methyl acetylene	1000.	lung	none	odor	lung-nar		rpt	1955
Methylal	1000.	chronic	odor	irr	lung-tox		rpt	1952
Methyl acrylate	10. T	lung	none	odor	irr-nar		rpt	1956
Methyl alcohol	200.	narcosis	none	odor	irr-nar-tox?		rpt	1943
Methyl bromide	20.	chronic	none	cns-odor	irr-tox	pyr	clin-rpt	1945
Methyl CELLOSOLVE	25.	chronic	none	odor-tox?	irr-nar-tox		clin-rpt	1947

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Methyl CELLOSOLVE acetate	25.	chronic	none	odor-tox	irr-nar-tox		est	1947
Methyl chloride	100.	chronic	none	odor	cns-nar	pyr	rpt	1947
Methyl chloroform	500.	narcosis	odor	nar	NAR	pyr	rpt	1953
Methyl cyclohexane	500.	narcosis	odor	nar	irr-NAR-tox		rpt	1947
Methyl cyclohexanol	100.	narcosis	irr-odor	nar-tox	IRR-NAR		rpt	1945
Methyl cyclohexanone	100.	narcosis	irr-odor		IRR-nar		rpt	1945
Methylene chloride	500.	chronic	none	odor	nar	pyr	rpt	1945
Methyl formate	100.	narcosis	none	irr-odor	nar		sgl	1947
Methyl isobutyl carbinol (methylamyl alcohol)	25.	narcosis	none	irr-odor	IRR-nar		hu-sgl	1954
Methyl mercaptan	50. T	lung	ODOR		eye-irr-lung?		sgl	1954
Molybdenum (soluble)	m-5.	chronic	none	irr-tox			rpt	1955
Molybdenum (insoluble)	m-15.	chronic	none	none	irr-tox		rpt	1955
Naphtha (coal tar)	200.	narcosis	irr-nar?-odor		NAR		est	1945
Naphtha (petroleum)	500.	narcosis	odor	irr-nar	NAR		est	1945
Nickel carbonyl	0.001	cancer-lung	none	none	none		ind-sgl	1954
Nicotine	m-0.5 T	chronic	none		tox	SKP	est	1956
Nitric acid	10. T	lung	irr	IRR	lung	BUR-EYE	est	1956
p-Nitroaniline	1.	chronic	none		tox	skp	est-ind	1954
Nitrobenzene	1.	chronic	none		tox	SKP	est	1947
Nitroethane	100.	acute	none	irr-odor	nar-tox		rpt	1947
Nitrogen dioxide	5.	lung	odor	irr	LUNG		ind-rpt	1945
Nitroglycerine	0.5	acute	head		tox	SKP	clin	1945
Nitromethane	100.	acute	none	irr-odor	nar-tox		rpt	1947
2-Nitropropane	50.	acute	nau-tox?		odor-tox		clin-sgl	1947
Nitrotoluene	5.	chronic	none	tox?	tox	SKP	est	1943
Octane	500.	narcosis	odor	irr-nar	NAR		est	1945
Ozone	0.1	lung	odor		irr-lung		rpt	1954
Parathion	m-0.1	acute	none		tox	SKP	clin-est	1953
Pentaborane	0.01 T	acute	none		none		rpt	1956
Pentachloronaphthalene	m-0.5	chronic	none	tox?	tox	acne	rpt	1945
Pentachlorophenol	m-0.5	acute	none		irr-tox	bur-skp	est	1947
Pentane	1000.	narcosis	odor	irr	nar		hu	1947
Pentanone (methyl propyl ketone)	200.	narcosis	irr-odor	IRR	nar		hu-sgl	1947
Perchloroethylene	200.	narcosis	odor	nar	irr-NAR	pyr	med-rpt	1953
Perchloromethyl mercaptan	0.1 T	lung	none		irr	bur-EYE	sgl	1954

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Phenol	5.	chronic	odor	irr	lung-tox	BUR-EYE-SKP	hu-rpt	1952
Phenylhydrazine	5.	chronic			cy-TOX	all-SKP	sgl	1954
Phosgene	1.	lung	none		LUNG-odor	bur-EYE	HU-rpt	1948
Phosphine	0.05	chronic	none		none		rpt	1947
Phosphorous (yellow)	m-0.1	chronic	none		tox?		ind	1947
Phosphorous pentachloride	m-1.	lung	none		irr-lung	bur-EYE	sgl	1947
Phosphorous pentasulfide	m-1.	lung	none		irr-lung		est	1947
Phosphorous trichloride	0.5	lung	none	irr	lung-odor	bur-EYE	sgl	1945
Picric acid	m-0.1	chronic	none	irr	tox	all-skp	clin	1954
Propyl acetate	200.	narcosis	irr-odor	IRR-nar	NAR		est-sgl	1945
Propyl alcohol (isopropanol)	400.	narcosis	irr-odor	nar	IRR-NAR		est-hu	1945
Propylene dichloride	75.	chronic	tox?	nar-odor	TOX	pyr	rpt	1947
Propylene imine	25.	acute-lung	odor		lung-TOX	bur-EYE-SKP	sgl	1955
Propyl ether	500.	narcosis	irr-odor	IRR	NAR		rpt	1945
Pyrethrum	m-2.	T lung	none		irr		rpt	1956
Pyridine	10.	chronic	cns?-odor		irr-tox		ind-med	1954
Quinone	0.1	eye pig	none	eye-pig	vis		clin	1955
Rotenone	m-5.	T lung	none		irr		est	1956
Selenium compounds, as Se	m-0.1	chronic	none		tox		clin-est	1947
Sodium hydroxide	m-2.	irr	irr		IRR	bur-EYE	ind	1954
Stibine	0.1	chronic-lung	none		lung?-tox?		sgl	1947
Stoddard solvent	500.	narcosis	odor	irr-nar	NAR		hu	1945
Strychnine	m-0.15	T acute	none		tox		est	1956
Styrene monomer	200.	narcosis	odor	irr	IRR-nar		hu-rpt	1947
Sulfur dioxide	10.	lung	irr-odor	IRR	lung		clin-hu	1943
Sulfur hexafluoride	1000.	asphyxia	none	none	none	pyr	sgl	1954
Sulfuric acid	m-1.	lung	irr		IRR-lung	bur-EYE	HU-ind-sgl	1948
Sulfur monochloride	1.	lung	none	irr	IRR-lung	bur-EYE	ind-sgl	1945
Sulfur pentafluoride	0.025	lung	none		irr-lung	bur-EYE	sgl	1954
TEDP	m-0.2	acute	none		tox		est	1954
Tellurium	m-0.1	chronic	none	none	irr-tox?		clin	1947
TEPP	m-0.05	acute	none		tox		est	1954
p-Tertiary butyl toluene	10.	chronic	odor		irr-nar-tox		hu-rpt	1955
Tetrachloroethane	5.	chronic	tox?	tox	odor-TOX	pyr	ind-sgl	1947
Tetrahydrofuran	200.	T narcosis	none	odor	irr		rpt	1956
Tetranitromethane	1.	acute	irr		tox	skp	rpt	1955
Tetryl	m-1.5	chronic	none	irr	tox	all-skp	clin	1943
Thallium (soluble)	m-0.15	T chronic					est	1956

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Proposed Year
Thiram	m-5.	T chronic	none				est-sgl	1956
Titanium dioxide	m-15.	lung	none		irr-lung		rpt	1954
Toluene	200.	narcosis	nar?-odor	irr	NAR		clin-hu-rpt	1943
o-Toluidine	5.	chronic	none	cy	CY-tox	SKP	sgl	1945
Trichloroethylene	200.	narcosis	nar-nau-odor		irr-NAR	pyr	ind-med-rpt	1948
Trichloronaphthalene	m-5.	chronic	none		tox	acne	rpt	1945
Trifluoromonobromomethane	1000.	narcosis	none	none	irr-nar	pyr	rpt	1955
Trinitrotoluene	m-1.5	chronic	tox?	tox?	irr-tox	skp	clin-rpt	1943
Turpentine	100.	narcosis	irr-odor	IRR-nar	NAR-tox		hu-rpt	1945
Uranium (soluble)	m-0.05	chronic	none		tox	rad	rpt	1953
Uranium (insoluble)	m-0.25	chronic	none		tox	rad	rpt	1953
Vanadium (V_2O_5 dust)	m-0.5	lung	none		lung		rpt	1954
Vanadium (V_2O_5 fume)	m-0.1	lung	none	lung			rpt	1954
Vinyl chloride	500.	narcosis	none		nar	pyr	sgl	1947
Warfarin	m-0.5	T chronic	none		tox		est	1956
Xylene	200.	narcosis	irr-nar?-odor	nar	NAR		hu	1943
Zinc oxide fume	m-15.	fume	fume	fume-irr	lung		HU	1943
Zirconium	m-5.	T lung	none		lung		rpt	1955

The most important effect of acetaldehyde inhalation is irritation of upper respiratory tract, bronchi and even lung. The 200 ppm threshold limit can be interpreted from human sensory data. It is sufficiently low to prevent lung injury.

Acetic acid. Sterner (1943) concludes 10 ppm is reasonably non-irritating on the basis of industrial experience. Patty (1948-9, p. 886) finds 800 to 1200 ppm intolerable. Smyth (1937-55) found inhalation of 16,000 ppm by rats for four hours killed one of six. The liquid causes severe corneal injury. Vigliani and Zurlo (1955) report workers exposed seven to 12 years to concentrations of 60 ppm, with one hour daily at 100 to 260 ppm, had no injury except slight irritation of the respiratory tract, stomach and skin. They regard 20 to 30 ppm as without danger.

The only important effect of acetic acid inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 10 ppm threshold limit can be interpreted from uncontrolled human sensory data. It is low enough to prevent lung injury.

Acetic anhydride. Henderson and Haggard (1943, p. 130) mention eye, nose and throat irritation and suggest that bronchial and lung injury are likely. Fairhall (1949, p. 203) considers it a lacrimator and finds systemic effects unlikely. McLaughlin (1946) discusses serious corneal injury from the liquid in industry. Smyth (1937-55) found rats inhaling 1000 ppm for four hours survived, but 2000 ppm was fatal. The liquid causes skin burns.

The only important effect of acetic anhydride inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 5 ppm threshold limit can be interpreted from analogy with acetic acid. In view of rat mortality from the two vapors, a lower value would be more consistent, although it is undoubtedly low enough to prevent lung injury.

Acetone. Nelson, Ege, Ross, Woodman and Silverman (1943) found slight eye,

nose and throat irritation with unacclimated subjects at 300 ppm, but 500 ppm was not objectionable. Henderson and Haggard (1943, p. 196) conclude death is anesthetic, with no organic injury below a narcotic level. Fairhall (1949, p. 205) concludes it causes narcosis, bronchial irritation and headache, but no chronic systemic effect. Haggard, Greenburg and Turner (1944) found human narcosis like that from ethyl alcohol. The highest concentration not causing narcotic impairment of coordination and judgment is 2110 ppm, which results in a blood level ⅓ that giving first alcoholic intoxication symptoms. Smyth's (1937-55) rats survived four hours at 32,000 ppm, died from 64,000 ppm. Vigliani and Zurlo (1955) found chronic respiratory tract irritation and dizziness in workers inhaling 1000 ppm three hours a day.

The most important effect of acetone inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from human sensory and physiological data. It is not low enough to prevent all narcotic symptoms.

Acrolein. Yant, Schrenk, Patty and Sayers (1930) found marked human eye, nose and throat irritation within five minutes at 1 ppm. Patty (1948-9, p. 936) concludes 0.25 ppm is moderately irritating. Henderson and Haggard (1943, p. 138) conclude the main attack is on the upper respiratory tract, but that a high concentration can cause lung edema. They report 10 ppm to be lethal in a short time. Systemic effects are not to be expected. Smyth (1937-55) found four hours inhalation of 8 ppm kills one of six rats and all die from 16 ppm. The liquid causes severe corneal injury and burns of the skin.

The only important effect of acrolein inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 0.5 ppm threshold limit can be interpreted from human sensory data. It is low enough to prevent lung edema.

Acrylonitrile. Dudley, Sweeney and Miller (1942) found repeated inhalation of 153 ppm injurious to animals, but believed the effect not chronic toxicity. Dudley and Neal (1942) concluded injury is due to formation of cyanide in the body. The 10 ppm hydrogen cyanide threshold limit is equivalent to 20 ppm acrylonitrile, if conversion is complete. Wilson (1944) in exposed workmen, found evidence of skin penetration and effects referable to liver injury. Smyth's (1937-55) rats survived four hours at 500 ppm but were killed at 1000 ppm.

The most important effect of acrylonitrile inhalation is acute poisoning, due to hydrolysis in the body to cyanide. The 20 ppm threshold limit can be interpreted from results of repeated animal inhalation studies and its relationship to the accepted 10 ppm threshold limit for hydrogen cyanide. It is low enough to prevent injury.

Aldrin. Princi and Spurbeck (1951) examined workers with one to three years exposure to 1 to 2.6 mg./cu.m. aldrin and related dusts, and found no clinical evidence of injury. McGee (1955), reviewing human cases and animal data, finds aldrin and lindane have similar actions. Acutely they increase central nervous system irritability, leading to convulsions. Chronically they injure the liver, with effects also on kidney, lung and nervous system, and they sensitize some skins. ACGIH (1954b) finds aldrin twice as toxic to animals acutely as lindane, and concludes half the threshold limit of the latter is tentatively appropriate.

The most important effect of aldrin inhalation is chronic poisoning centering in the liver. The 0.25 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It is low enough to prevent injury.

Allyl alcohol. McCord (1932) found some human irritation at 5 ppm. The review by von Oettingen (1943, p. 138) shows cats die during 30 seven-hour inhalations of 50 ppm, with pulmonary edema, gastroenteritis, hematuria and nephritis. Smyth (1937-55) found rats survive one hour at 500 ppm, but die from 1000 ppm. The vapors irritate eye and nose, but not sufficiently so to prevent exposure to a concentration which temporarily blinded one man through delayed corneal necrosis. Chronic toxicity is not to be expected, but skin penetration is dangerous, and skin contact causes burns when evaporation is prevented.

The most important effect of allyl alcohol inhalation is irritation, manifest as disabling corneal injury and pulmonary edema, with non-progressive organic effects somewhat less important and narcosis overshadowed. The 5 ppm threshold limit can be

interpreted from a report of human irritation and results of repeated animal inhalation. It is probably low enough to prevent injury, but may be slightly irritating to some.

Allyl chloride. Adams, Spencer and Irish (1940) found rats survive three hours inhalation of 290 ppm, while eight hours killed all. Injury was chiefly in the lung, with some kidney effects. Narcosis was weak but mucous membrane irritation was prominent.

The most important effect of allyl chloride inhalation is irritation of the upper respiratory tract, bronchi and lung. The 5 ppm tentative threshold limit can be interpreted from single inhalations by animals and by analogy with chloroprene. It appears low enough to prevent injury.

Allyl propyl disulfide. Feiner, Burke and Baliff (1946) surveyed an onion dehydrating plant and found pronounced irritation of eye, nose and throat at 3.4 ppm onion oil calculated as this disulfide, with some irritation at 2 ppm. Acclimitization was evident.

The most important effect of allyl propyl disulfide inhalation is irritation of eye, upper respiratory tract and lung. The 2 ppm threshold limit can be interpreted from the results of complaints from exposed workmen. It appears low enough to prevent injury.

Ammate. ACGIH (1954b) concludes the single dose LD_{50} for animals of 2000 mg./kg. justifies no more control on inhalation than is required for a non-toxic nuisance dust.

The most important effect of ammate dust inhalation is the low grade irritation of a substantially inert dust. The 15 mg./cu.m. threshold limit can be interpreted only by analogy. It appears low enough to prevent injury.

Ammonia. Lehmann (1886) suggested 100 ppm is tolerable, and subsequent industrial experience has been favorable. Henderson and Haggard (1943, p. 125) note incapacitating temporary blindness, and respiratory arrest from a high concentration. They state 53 ppm is the least amount smelled, but Smyth (1937-55) found 1 ppm detected and identified by 10 subjects. They give 408 ppm as irritating to the throat, 698 ppm irritating to the eye, 2500 to 6500 ppm dangerous to life in 30 minutes. Fairhall (1949, p. 20) notes eye and upper respiratory tract irritation, salivation, bronchial irritation and lung edema, but no chronic systemic effect. Elkins (1950, p. 84) found 55 ppm not objectionable in industry but 125 ppm irritating. Silverman, Whittenberger and Muller (1949) found 500 ppm stimulated human respiration, irritated eye and throat, caused lacrimation. Smyth (1937-55) found rats survive four hours at 2000 ppm, die at 4000 ppm. Solutions irritate the skin, erode mucous membrane and severely injure the cornea. Vigliani and Zurlo (1955) in workers inhaling 100 ppm, found irritation of the respiratory tract and conjunctiva. Even 20 ppm caused complaints until workers became hardened.

The most important effect of inhalation of ammonia gas is respiratory tract irritation, with lung edema or respiratory arrest the maximum injury. The 100 ppm threshold limit can be interpreted from human sensory and physiological data. It is low enough to prevent injury.

Amyl acetate. Patty, Yant and Schrenk (1936) found 2000 ppm does not injure guinea pigs in several hours, while a concentration killing in 60 minutes can not be obtained. Symptoms consisted of eye and nose irritation and narcosis. Nelson, Ege, Ross, Woodman and Silverman (1943) found slight throat irritation in unacclimated subjects at 100 ppm, mild eye and nose sensation and severe throat irritation at 200 ppm. Smyth (1937-55) with rats inhaling substantially saturated vapors found anesthesia in two hours and death in eight hours. Chronic toxicity is not to be expected.

The most important effect of amyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted from human sensory data and single inhalations by animals. It is low enough to prevent definite narcosis.

Amyl alcohol (isoamyl alcohol.) Nelson, Ege, Ross, Woodman and Silverman (1943) found slight throat irritation in unacclimated subjects at 100 ppm, and objectionable eye, nose and throat irritation at higher concentrations. Haggard, Miller and Greenberg (1945) found the toxicity 12 times that of ethyl alcohol for anesthetic death. No chronic systemic toxicity is to be expected. Smyth (1937-55) found rats not

killed by eight hours at 2000 ppm, close to saturation.

The most important effect of amyl alcohol inhalation is narcosis. The 100 ppm threshold limit can be interpreted from human sensory data and analogy with butyl alcohol. It is low enough to prevent significant narcosis, but not to prevent slight irritation.

Aniline. Henderson and Haggard (1943, p. 227) conclude 7 to 25 ppm gives slight symptoms in several hours, 100 to 160 ppm for one hour causes serious disturbance. Aniline is a chemical asphyxiant, causing methemoglobin cyanosis through its metabolite, p-aminophenol. This can lead to anemia, but death from a single exposure is due to central nervous effects leading to respiratory paralysis. Skin penetration is more an industrial hazard than inhalation. Smyth (1937-55) found 340 ppm, substantial saturation, did not kill rats in two hours but was fatal in four, with their hemoglobin 54% converted to methemoglobin. Oberst, Hackley and Comstock (1956) found repeated inhalation of 5 ppm caused methemoglobinemia in rats but not any symptoms in dogs. They explained the difference in response by the fact that rats breathe three times as much air per unit time as do dogs, hence absorbed more aniline.

The most important effect of aniline inhalation is acute poisoning, in which cyanosis is evident but not of major importance. The 5 ppm threshold limit can be interpreted from results of repeated animal inhalations. It appears low enough to prevent injury.

Antimony. Bradley and Fredrick (1941) administered various antimony compounds orally and intraperitoneally to rats. They concluded it is more toxic than lead, but is not stored. The most important effect was on the heart muscle, and they advised that exposed workmen should be followed electrocardiographically. Dernehl, Nau and Sweets (1945) exposed guinea pigs to antimony oxide of one micron diameter at 45 mg./cu.m., three hours a day for several months. One-sixth died of pneumonitis with severe liver injury and white blood cell changes, but hearts remained normal. Electrocardigrams on the animals and on a few industrially exposed workmen were normal. Brieger, Senisch, Stasney and Piatnek (1945) in an industrial operation where antimony trisulfide concentrations ranged from 0.58 to 5.5 mg./cu.m., found abnormalities in blood pressure, electrocardiographic changes and two deaths from chronic thrombosis.

The most important effects of inhalation of antimony dust are chronic poisoning marked by electrocardiographic changes, pneumonitis and liver injury. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated inhalations and examination of workmen. It appears low enough to prevent injury.

ANTU (alphanaphthylthiourea). McClosky and Smith (1945) found considerable species differences in acute oral toxicity. The LD_{50} for rats, the most susceptible species, was about 0.03 gm./kg. Death was due to pleural efusion. Repeated doses caused liver injury. Fitzhugh and Nelson (1947) found rats are not affected by 50 ppm in their diet over a two-year period. Tolerance developed.

The most important effect of ANTU inhalation is chronic poisoning centering in the liver. The 0.3 mg./cu.m. tentative threshhold limit can be interpreted from the results of repeated oral doses to rats. It corresponds to a maximum daily absorption of three milligrams, about 0.05 mg./kg. This appears low enough to prevent injury.

Arsenic. The earlier limit of 0.15 mg./cu.m. was based upon supposed quantitative similarity to lead. Watrous and McCaughey (1945) found workers exposed to 0.007 to 0.60 mg./cu.m. showed no symptoms. Chronic arsenic poisoning causes varied symptoms (I.L.O., 1930, I, p. 161): digestive disturbance, hyperemia, skin eruption, pigmentation, keratosis, epithelial cancer, polyneuritis, cirrhosis, confusion, delirium, irritation of eyes and respiratory tract. Arsenic is stored in the body, but not to the extent of lead.

The most important effect of inhalation of arsenic compounds is chronic poisoning. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of examinations of exposed workmen. It appears low enough to prevent injury.

Arsine. Henderson and Haggard (1943, p. 241) describe acute arsine poisoning as due to hemolysis of red blood cells with resulting anemia and kidney damage, and lung

edema. They state 250 ppm for 30 minutes is fatal, and 3 to 10 ppm can cause symptoms in a few hours. Nau (1948) reported an industrial episode showing the earlier limit of 1 ppm was too high, and Elkins (1950, p. 67) reported briefly on a non-fatal case at a level of about 0.5 ppm.

The most important effect of arsine inhalation is acute poisoning, largely lung edema. The 0.05 ppm threshold limit can be interpreted from the results of industrial experience. It appears to be low enough to prevent injury.

Barium (soluble compounds). Fairhall (1949, p. 32) records the fatal dose of soluble barium compounds as 0.8 to 0.9 grams with gastro-intestinal disturbance the chief symptom. He notes bronchial irritation from barium carbonate dust, and depilatory action of barium sulfide.

The most important effect of inhalation of soluble barium compounds is bronchial irritation, with acute poisoning possible. The 0.5 mg./cu.m. threshold limit can be interpreted only by analogy with antimony. It appears low enough to prevent injury.

Benzene. Winslow (1927) first proposed a limit of 100 ppm, based on extensive examination of exposed workmen and animal inhalation. He recognized that chronic poisoning would develop in some at this concentration, but believed it would progress slowly enough to be detected by periodic medical examinations, and arrested by removal from exposure. Acute benzene poisoning is fatal anesthesia, and chronic poisoning is primarily injury to the bone marrow. Benzene is particularly insidious because its effects can progress to a fatal outcome after all exposure ceases. Even brief inhalation of a high non-anesthetic concentration can be fatal. Patty (1948-9, p. 757) states that 100 ppm has only a faint odor. Elkins (1950, p. 228) investigated a fatal case whose exposure he was convinced had been only to 40 to 80 ppm.

The most important effect of benzene inhalation is chronic poisoning centering in the bone marrow. The 35 ppm threshold limit can be interpreted from extensive examination of exposed workmen, and was quantitatively defined by one fatal case. It appears low enough to prevent the development of irreversible poisoning.

Benzyl chloride. This is a potent lacrimator, irritating to eye, nose and throat, and capable of causing lung edema. Flury and Zernik (1931, p. 538) conclude 170 ppm is dangerous to cats in eight hours and 16 ppm intolerable to man in one minute. It may be inferred that the liquid causes severe corneal injury.

The only important effect of benzyl chloride inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 1 ppm threshold limit can be interpreted from older human sensory data. It is undoubtedly low enough to prevent lung injury.

Bromine. Flury and Zernik (1931) quote Lehmann that 0.75 ppm in a workroom caused no symptoms in six hours. Henderson and Haggard (1943, p. 133) state bromine acts as a respiratory irritant leading to lung edema. They state 40 to 60 ppm is dangerous on short inhalation, and 4 ppm allowable for 30 to 60 minutes. Elkins (1950, p. 87) found 1 ppm excessively irritating. Severe burns of skin and cornea result from the liquid. Patty (1948-9, p. 554) concludes 0.3 ppm is not objectionably irritating.

The most important effect of inhalation of bromine vapor is respiratory tract irritation, with lung edema the maximum effect. The 1 ppm threshold limit can be interpreted from industrial experience. It appears low enough to prevent injury.

Butadiene. Von Oettingen (1940) quotes repeated animal exposures at 64,000 ppm which caused bronchial and lung irritation and some hyperplasia of bone marrow. Carpenter, Shaffer, Weil and Smyth (1944) found animals not affected by repeated inhalation of 2300 ppm, while 6700 ppm slightly retarded growth and there were minor liver effects. Two humans found psychomotor effects of early narcosis from 8000 ppm, equivalent to those from 200 ppm toluene.

The most important effect of butadiene vapor inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from results of repeated animal inhalation and single human inhalation. It is low enough to prevent any degree of narcosis.

Butanone (methyl ethyl ketone). Patty, Schrenk and Yant (1935) found guinea pigs tolerated 3000 ppm for several hours, and men found it irritating to nose and eyes. Nelson, Ege, Ross, Woodman and Silverman

(1943) found slight throat irritation in unacclimated subjects at 100 ppm, irritation of eyes at 200 ppm and objectionable irritation at 300 ppm. Elkins (1950, p. 118) found complaints of nausea at 500 ppm, irritation at 300 ppm, but no ill effects at 700 ppm. Smyth (1937-55) found rats survived two hours at 2000 ppm but 4000 ppm killed four of six. He found that an episode of industrial eye injuries during inhalation of butanone was caused by an unsaturated ketone impurity accidently present.

The most important effect of butanone inhalation is narcosis. The 250 ppm threshold limit can be interpreted from human sensory data. It appears low enough to prevent definite narcosis.

Butyl acetate. Sayers, Schrenk and Patty (1936) found guinea pigs are not affected by several hours inhalation of 3300 ppm. Nelson, Ege, Ross, Woodman and Silverman (1943) found throat irritation in unacclimated subjects at 200 ppm, severe at 300 ppm. Henderson and Haggard (1943, p. 222) conclude the ester shows no chronic toxicity. Smyth (1937-55) found rats inhaling substantially saturated vapors are not killed in four hours, but died within an eight-hour inhalation period.

The most important effect of butyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted from human sensory response and single inhalations by animals. It is low enough to prevent definite narcosis.

Butyl alcohol (n-butanol). Tabershaw, Fahy and Skinner (1944) reported eye inflammation in workmen above 50 ppm, but no systemic effects below 100 ppm. Sterner, Crouch, Brockmyre and Cusack (1949) followed workmen for 10 years with butyl alcohol concentrations held to 100 ppm, and for a briefer period to 200 ppm. Neither irritation nor systemic effects were found at 100 ppm, but there was some eye irritation at 200 ppm. Smyth (1937-55) found rats are not killed in four hours at 8000 ppm.

The most important effect of butyl alcohol inhalation is narcosis. The 100 ppm threshold limit can be interpreted from an extensive study of workmen under conditions of known peak exposure. No narcotic or irritative effects are to be anticipated.

Butyl amine. Hanzlik (1923) reported central nervous stimulation, convulsions, then depression and narcotic death with pulmonary edema. Smyth (1937-55) found rats survive four hours at 2000 ppm but die from 4000 ppm. Injury to skin and cornea from the liquid is severe. Unreported industrial experience suggests skin injury is the greatest practical hazard. ACGIH (1955b) cites unpublished industrial experience that levels above 5 ppm tend to be irritating.

The most important effect of butyl amine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 5 ppm threshold limit can be interpreted from analogy with ethyl amine. It is probably low enough to prevent injury.

Butyl CELLOSOLVE (2-butoxyethanol). Werner, Nawrocki, Mitchell, Miller and von Oettingen (1943) found 300 to 400 ppm, in repeated inhalation produced only small effects on rats, particularly on the blood picture. Werner, Mitchell, Miller and von Oettingen (1943a, b) reporting on single inhalations by rats and repeated by dogs, make it clear that the butyl ether produces somewhat greater blood cell changes than do the methyl or ethyl ethers. They also found hemoglobinuria, with lung, liver and kidney changes. Smyth (1937-55) found in rats fractional mortality from as little as 500 ppm inhaled eight hours, with hematuria a prominent symptom. The liquid penetrates the skin readily and is sufficiently toxic so that this is dangerous.

The most important effect of butyl CELLOSOLVE inhalation is chronic poisoning, centering in the blood cells and kidney. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. Based on reports from simultaneous studies of CELLOSOLVE and butyl CELLOSOLVE it is obvious that the threshold limit for the latter should be lower than for the former if equal degrees of protection are to be attained.

Butyl mercaptan. Fieldner, et al. (1931) reports 733 ppm to be lethal to dogs in 30 minutes, indicating 20 times the acute toxicity of ethyl mercaptan. Effects like those of hydrogen sulfide are to be expected.

The most important effect of butyl mercaptan is eye and respiratory tract irritation. The 10 ppm tentative threshold limit can be interpreted from the results of limited single inhalations by animals and analogy with hydrogen sulfide. It appears low

enough to prevent injury and eye irritation.

Cadmium oxide fume. Prodan (1932) on the basis of animal experiment concluded it is as toxic as lead. Spolyar, Keppler and Porter (1944) reported on serious poisoning and fatalities from industrial exposure to the fume. Fairhall (1949, p. 45) concludes inhalation causes bronchial irritation and pneumonitis, while ingestion produces gastro-intestinal disturbances.

The most important effect of inhalation of cadmium oxide fume is severe lung injury, with systemic poisoning less important. The 0.1 mg./cu.m. threshold limit cannot be interpreted quantitatively, but it appears low enough to prevent injury.

Calcium Arsenate. ACGIH (1954b) bases the tentative threshold limit upon a rat oral LD_{50} of 100 mg./kg., and blind litters in rats fed 5 mg./kg. for 45 days.

The most important effect of inhalation of calcium arsenate dust is chronic arsenic poisoning, with bronchial irritation less important. The 0.1 mg./cu.m. tentative threshold limit should be interpreted from the threshold limit for arsenic dusts. Since calcium arsenate is 20% arsenic, a threshold limit of 2.5 mg./cu.m. would be consistent with the accepted limit for arsenic dusts.

Carbon dioxide. Flury and Zernik (1931) quote Lehman-Hess to the effect that 5500 ppm causes no noticeable symptoms in six hours. Aero Medical Association (1953, p. 52) considers the gas as weakly narcotic, 30,000 ppm increasing respiration by 90%, increasing pulse and blood pressure, and decreasing acuity of hearing. Subjective symptoms arise above this level, 50,000 ppm for 30 minutes giving the first signs of intoxication, and 70,000 to 100,000 ppm causing unconsciousness in a few minutes.

The most important effect of carbon dioxide inhalation is asphyxia at very high concentrations. The 5000 ppm threshold limit can be interpreted from the results of extensive human experiments. It is low enough to prevent noticeable effects.

Carbon disulfide. Wiley, Hueper and von Oettingen (1936) found repeated inhalation of 30 ppm has no significant effect on animals. Barthelemy (1939) found no injuries to rayon workmen when concentrations were kept below 30 ppm. In single inhalations, it is markedly narcotic, and in repeated exposure the effects may be neurological, not recognized by the victim. Henderson and Haggard (1943, p. 223) state that 480 to 1600 ppm is the maximum breathable for one hour without serious disturbance.

The most important effect of carbon disulfide inhalation is chronic poisoning with central nervous system effects. The 20 ppm threshold limit can be interpreted from the results of repeated animal inhalations and examination of exposed workmen. It is low enough to prevent injury.

Carbon monoxide. Henderson, Haggard, Teague, Prince and Wunderlich (1931) suggested a limit of 100 ppm on the basis of extensive human experiment. Sayers, Yant, Levy and Fulton (1929) showed 200 ppm caused slight symptoms in humans. Sievers, Edwards, Murray and Schrenk (1942) found 70 ppm over a 13-year period had not affected health. Carbon monoxide is a chemical asphyxiant, acting by combining with hemoglobin. Henderson and Haggard (1943, p. 167) define its effects in terms of the product of time and concentration, 100 ppm for three hours producing no effect; for six hours, a just appreciable effect; for nine hours, headache and nausea; and for 15 hours, danger. One hour at 4000 ppm may be fatal. Vigliani and Zurlo (1955) studying 100 workers found no injury to health at 100 ppm for eight hours every day. In the United States chronic poisoning is not considered a reality.

The most important effect of carbon monoxide inhalation is chemical asphyxia, reducing the oxygen carrying power of the blood. The 100 ppm threshold limit can be interpreted from the results of extensive human experiment and examination of exposed workman. It will prevent injury, but will allow a recognizable effect if inhaled for eight hours.

Carbon tetrachloride. Elkins (1950, p. 229) on the basis of industrial experience, concluded the earlier figure of 100 ppm was too high and suggested 40 ppm. Adams, Spencer, Rowe, McCollister and Irish (1952) in extensive animal studies, found some effect on the liver in some species at all concentrations above 5 ppm. Smyth (1937-55) found rats survive eight hours at 3000 ppm, but 8000 ppm is fatal. Human anesthesia, or near anesthesia, is usually fatal from

kidney injury, while early narcosis occurs at a low concentration. Chronic toxicity is chiefly marked by liver injury.

The most important effect of carbon tetrachloride inhalation is chronic toxicity centering in the liver. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalations and industrial experience. It is low enough to prevent irreversible injury, but perhaps it will allow minor injury.

CELLOSOLVE *(2-ethoxyethanol)*. Werner, Nawrocki, Mitchell, Miller and von Oettingen (1943) found rats repeatedly inhaling 300 to 400 ppm showed small but measurable blood cell effects. Werner, Mitchell, Miller and von Oettingen (1943b) found dogs inhaling 800 ppm repeatedly developed small blood cell effects. Smyth (1937-55) found rats survive four hours at 2000 ppm, but half are killed by 4000 and all are killed by eight hours at 4000 ppm, close to saturation. Death is marked by severe kidney damage.

The most important effect of CELLOSOLVE inhalation is chronic poisoning centering in the red blood cells. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. It appears to be low enough to prevent injury. There are no data to judge the degree of eye and nose irritation it allows.

CELLOSOLVE *acetate (2-ethoxyethyl acetate)*. Smyth (1937-55) found in dogs after 120 seven-hour inhalations of 600 ppm, only a small increase in bromosulfalein retention, with eye and nose irritation. Rats survive 1500 ppm (close to saturation) for four hours, but two of six die after eight hours. It is easily hydrolyzed to CELLOSOLVE and acetic acid. Systemic injury should follow closely that of CELLOSOLVE, but respiratory tract irritation is somewhat greater.

The most important effect of CELLOSOLVE acetate is chronic poisoning due to hydrolysis to CELLOSOLVE. The 100 ppm threshold limit can be interpreted from analogy with CELLOSOLVE. It appears low enough to prevent injury. There are no data to judge the degree of eye and nose irritation it allows.

CHLORDANE. Princi and Spurbeck (1951) quote animal data indicating effects are principally neurological, with liver and kidney injury and pulmonary irritation. They found workers for three years with exposures of the order of 5 mg./cu.m. showed no clinical evidence of effect. Alvarez and Hyman (1953) examined men in another producing plant with up to five years exposure and found no effects, but concentrations were not measured. Ingle (1953) shows that early reports of inhalation injury in animals were due to a volatile unreacted intermediate in the early product, and that 14 days continuous inhalation of saturated air does not injure mice. ACGIH (1954b) bases its tentative threshold limit on a rat oral LD_{50} of 590 mg./kg.

The most important effect of chlordane inhalation is chronic poisoning centering in the liver. The 2 mg./cu.m. threshold limit can be interpreted from the results of examinations of exposed workmen. It is low enough to prevent injury.

Chlorinated camphene, 60% (toxaphene). Lackey (1949) found an oral dose of 10 mg./kg. caused convulsions in dogs while 15 mg./kg. was fatal. A daily dose of 4 mg./kg. for 106 days was not fatal, but at times convulsions were seen. Liver and kidney changes resulted.

The most important effect of chlorinated camphene is chronic poisoning centering in the liver. The 0.5 mg./cu.m. tentative threshold limit can be interpreted from the results of single and repeated oral doses, and by analogy with the similar but less toxic DDT. It appears low enough to prevent injury.

Chlorinated diphenyl oxide. After extensive inhalation studies with rats, Drinker (1949) concluded that 0.5 mg./cu.m. is a permissible concentration which will not lead to systemic injury. Liver injury is the effect of chronic poisoning. Smyth (1937-55) found the material penetrates the skin, and repeated contact leads to chloracne.

The most important effect of chlorinated diphenyl oxide inhalation is chronic poisoning centering in the liver. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Chlorine. Sklyanskaya and Rappaport (1935) found lung injuries and increased incidence of pneumonia in guinea pigs repeatedly inhaling 0.7 to 1.7 ppm. Fairhall (1950, p. 52) states it irritates eyes and nose, and may cause fatal lung irritation. Inhalation of 1000 ppm is rapidly fatal, 40

to 60 ppm may lead to pneumonitis and lung edema, 30 ppm causes coughing, 15 ppm throat irritation and 3.5 ppm can be smelled. Patty (1948-9, p. 547) concludes 1 to 2 ppm is tolerable, 3 to 6 ppm irritating.

The most important effect of inhalation of chlorine gas is respiratory tract irritation, with lung edema the maximum effect. The 1 ppm threshold limit can be interpreted from repeated animal inhalation and human sensory data. It is low enough to prevent injury.

Chlorine trifluoride. Horn and Weir (1955) found it an extremely active irritant. Rats are killed in 40 minutes at 96 ppm, while rats and dogs inhaling 5 ppm repeatedly are severely injured. Pneumonia was increased and there was respiratory difficulty in all. The vapors injure the cornea. Horn and Weir (1956) found repeated inhalation of 1.17 ppm by rats and dogs injured only by increased incidence of pneumonia.

The most important effect of inhalation of chlorine trifluoride gas is respiratory tract irritation, with lung edema the maximum effect. The 0.1 ppm threshold limit can be interpreted from repeated animal inhalation. It appears low enough to prevent injury.

Chlorobenzene. Fairhall (1949, p. 260) concludes it is somewhat more toxic than benzene, but finds no evidence of hematopoetic effect.

The most important effect of chlorobenzene inhalation is narcosis. The 75 ppm threshold limit can be interpreted only as a rough estimate. By comparison with other chlorinated hydrocarbons, it appears low enough to prevent injury.

Chlorobromomethane. Svirbely, Highman, Alford and von Oettingen (1947) found 3000 ppm to be the LC_{50} for mice in eight-hour inhalations. Exposures of rats, rabbits and dogs to 1000 ppm seven hours a day five days a week for fourteen weeks were without effect. Non-progressive liver injury was found from single inhalations, but liver and kidney remained normal during the repeated inhalations. Comstock et al. (1952) found light narcosis in rats and mice inhaling 3000 ppm for 10 to 15 minutes, and about 30,000 ppm was fatal within 15 minutes. Pulmonary edema was present in animals dying.

The most important effect of inhalation of chlorobromomethane is narcosis with non-progressive effects on liver and kidney. The 400 ppm tentative threshold limit can be interpreted from the results of repeated inhalations with animals. It appears low enough to prevent injury.

Chlorodiphenyl (42% chlorine). After extensive inhalation studies with rats, Drinker (1939) concluded that 10 mg./cu.m. of a sample 68% chlorine, but free from chlorinated diphenyl benzene, would lead to no systemic injury, but that the presence of chlorinated diphenyl benzene reduced the permissible limit to 0.5 mg./cu.m. Smyth (1937-55) found the material penetrates the skin, and repeated contact leads to chloracne. Treon, Cleveland, Cappel and Atchley (1956) reported that inhalation of 8.6 mg./cu.m. for 24 seven-hour periods did not affect four species of animals, and 1.9 mg./cu.m. for 150 periods also was without effect.

The most important effect of chlorodiphenyl inhalation is chronic poisoning, centering in the liver. The 1 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Chlorodiphenyl, 54% chlorine. Treon, Cleveland, Cappel and Atchley (1956) reported that repeated inhalation over a seven-month period of 1.5 mg./cu.m. caused some minor liver injury in four species of rodents.

The most important effect of inhalation of chlorodiphenyl (54% chlorine) is chronic toxicity centering in the liver. The 0.5 mg./cu.m. tentative threshold limit can be interpreted from the results of repeated inhalation by animals. It appears to be slightly below an injurious concentration.

Chloroform. Fairhall (1949, p. 264) concludes it acts much like carbon tetrachloride, and that anesthetic use has led to liver injury, but regression is more likely than with carbon tetrachloride. A concentration of 4000 ppm causes slight symptoms after several hours exposure. Patty (1948-9, p. 793) concludes the least concentration smelled is 200 to 300 ppm. Smyth (1937-55) found one of six rats die from four hours at 4000 ppm, and all from 8000 ppm.

The most important effect of inhalation of chloroform is chronic poisoning centering in the liver. The 100 ppm threshold limit can be interpreted from the results of single

animal inhalations and human sensory response. It appears low enough to prevent injury, but in view of changes in ideas about carbon tetrachloride, new data are desirable.

1-Chloro-1-nitropropane. Machle, Scott, Treon, Heyroth and Kitzmiller (1945) exposed animals for six hours to 400 ppm. Effects were chiefly irritation of eye, nose bronchi and lung, with some injury to liver, kidney and vascular system, and 25% died. Simultaneous repeated studies on 1,1-dichloro-1-nitroethane showed little cumulative action.

The most important effect of 1-chloro-1-nitroethane is lung injury. The 20 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears low enough to prevent injury.

Chloropicrin. Fairhall (1949) concludes it is an irritant gas, producing bronchial and lung injury. He concludes 20 ppm produces lesions within one to two minutes and that 4 ppm will incapacitate a man.

The most important effect of chloropicrin is respiratory tract irritation with lung injury probable. The 1 ppm tentative threshold limit can be interpreted from summaries of studies of its use as a war gas. It appears low enough to prevent serious injury.

Chloroprene (2-chlorobutadiene). Von Oettingen, Hueper, Deichmann-Gruebler and Wiley (1936) made repeated inhalation studies with animals. A concentration of 83 ppm caused some respiratory irritation, central nervous system depression, and effects on liver, kidney and vascular system. Skin penetration was dangerous.

The most important effect of chloroprene inhalation is chronic poisoning, centering in the liver. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Chromic acid. Bloomfield and Blum (1928) and Riley and Goldman (1937) demonstrated in industrial surveys that perforation of the nasal septum and other upper respiratory tract effects of chromic acid mists are not found at a concentration of about 0.1 mg./cu.m. Other effects are not produced. Vigliani and Zurlo (1955) found ulcerated nasal septum, inflamed larynx, chronic bronchitis and two respiratory tract cancers among 150 workers exposed to 0.11 to 0.15 mg./cu.m.

The most important effect of inhalation of chromic acid aerosol is upper respiratory tract irritation, leading to perforation of the nasal septum. The 0.1 mg./cu.m. threshold limit can be interpreted from studies of exposed workmen. It is low enough to prevent injury.

CRAG herbicide. ACGIH (1954b) suggests on the basis of a general low toxicity and a rat oral LD_{50} of 1500 mg./kg., that 10 mg./cu.m. is a fit limit. Smyth (1937-55) found death from a single oral dose due to respiratory paralysis with injuy to liver and kidney. Rats are not affected by 0.02% in their diet for two years, and 0.06% causes minor effects on liver and kidney.

The most important effect of CRAG herbicide inhalation is chronic poisoning centering in the liver. The 15 mg./cu.m. threshold limit can be interpreted from the results of single and repeated oral doses to animals. It appears low enough to prevent injury.

Cresol. Fairhall (1949, p. 271) concludes effects and practical hazards are like those of phenol. Smyth (1937-55) found rats survive eight hours inhalation of vapors substantially saturated at room temperature. The liquid penetrates the skin to a dangerous extent, and causes severe skin and corneal injury. The odor appears to be somewhat more intense than that of phenol.

Although cresol vapors are odorous and irritating, their major effect is chronic systemic poisoning. The 5 ppm threshold limit can be interpreted from analogy with phenol. It appears to be low enough to prevent chronic poisoning.

Cyanide as CN. To the extent that cyanide dusts dissolve, their toxicity is that of hydrogen cyanide, with some added local irritation due to hydrolysis on moist tissue. Cyanide dust equivalent of the 10 ppm threshold limit for hydrogen cyanide is 11 mg./cu.m. The 5 mg./cu.m. threshold limit is about half that for hydrogen cyanide, and hence is conservative.

Cyclohexane. Treon, Crutchfield and Kitzmiller (1943) found minor liver and kidney changes in animals repeatedly inhaling 786 ppm, none at 434 ppm. Fairhall (1949, p. 273) concludes acute poisoning is anesthesia and that repeated inhalation causes no hematopoetic changes. Patty

(1948-9, p. 769) reports 300 ppm has no distinct odor or irritation.

The most important effect of cyclohexane inhalation is narcosis, with non-progressive organic effects. The 400 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent definite narcosis.

Cyclohexanol. Treon, Crutchfield and Kitzmiller (1943) reported that repeated inhalation of 693 ppm caused minimal pathological changes in a monkey, and 145 ppm in the livers and kidneys of rabbits. Nelson, Ege, Ross, Woodman and Silverman (1943) found 100 ppm causes objectionable eye, nose and throat irritation in unacclimated subjects. Smyth (1937-55) was unable to kill rats by eight hours inhalation of substantially saturated vapors.

The most important effect of cyclohexanol inhalation is narcosis, with non-progressive organic effects less prominent. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations and human sensory data. It is low enough to prevent significant narcosis or injury, but not to prevent irritation.

Cyclohexanone. Treon, Crutchfield and Kitzmiller (1943) in animal experiments found only narcosis and irritation. Nelson, Ege, Ross, Woodman and Silverman (1943) found 50 ppm caused objectionable eye, nose and throat irritation in unacclimated subjects. Smyth (1937-55) did not kill rats by four hours at 4000 ppm, but 8000 ppm caused anesthetic death.

The most important effect of cyclohexanone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from repeated animal inhalations and human sensory response. It is probably low enough to prevent definite narcosis.

Cyclohexene. Fairhall (1949, p. 279) concludes that 9000 ppm causes mild narcosis in animals, while 13,500 to 15,000 ppm gives anesthetic death. This indicates a toxicity greater than that of cyclohexane.

The most important effect of cyclohexene inhalation is narcosis. The 400 ppm threshold limit can be interpreted from results of single animal inhalations. It is low enough to prevent definite narcosis.

Cyclopropane. Fairhall (1949, p. 280) reviews reports of experience in surgical anesthesia and concludes there is no toxic hazard in industrial use. The most important effect of cyclopropane gas inhalation is narcosis. The 400 ppm threshold limit can be interpreted from analogy with cyclopentane, and by human surgical use. It is probably low enough to prevent definite narcosis.

2,4-D. Rowe and Hymas (1954) conclude that it has a low degree of chronicity, and the acute LD_{50} values range from 300 to 1000 mg./kg. for various species.

The most important effect of 2,4-D inhalation is chronic poisoning centering in the liver. The 10 mg./cu.m. threshold limit can be interpreted from the results of single and repeated oral doses to animals. It appears low enough to prevent injury.

DDT. Barnes (1953) finds no incidence of illness among workers using it throughout the world. Poisoning from accidental ingestion is marked by abdominal pain, vomiting, dizziness and weakness. Long repeated doses of 25 to 50 mg./kg. are required to poison animals, although man is probably more sensitive.

The most important effect of DDT inhalation is chronic poisoning centering in the liver. The 1 mg./cu.m. tentative threshold limit can be interpreted from the results of repeated oral doses to animals. It appears low enough to prevent injury.

Decaborane. Svirbely (1954a,b) found the LC_{50} for mice inhaling vapors for four hours to be 25.7 ppm. Symptoms included central nervous excitability and corneal opacity. Six-hour inhalations of 20 ppm by rats, repeated 20 times, killed some with fatty livers and central nervous excitability. Comstock and Oberts (1953) report that the median detectable odor is 0.35 mg./cu.m. (0.07 ppm), described as foul or chocolate-like.

The most important effect of decaborane inhalation is acute toxicity involving the central nervous system, with liver injury less important. The 0.05 ppm tentative threshold limit can be interpreted from limited animal inhalations. It appears to allow an adequate margin of safety.

Diacetone alcohol. Von Oettingen (1943, p. 138) reports animals at 2100 ppm are restless, irritation and kidney effects are noted. He concludes it is twice as toxic as acetone. Silverman, Schulte and First (1946) found 100 ppm irritating to eyes, nose and throat but not intolerable in un-

acclimated subjects. Smyth (1937-55) found 1500 ppm, approaching saturation, did not kill rats in eight hours.

The most important effect of diacetone alcohol inhalation is narcosis. The 50 ppm threshold limit can be interpreted from single animal inhalations and human response. It appears low enough to prevent definite narcosis.

Diborane. Rozendaal (1951) reported on five human injuries from inhalation of diborane and other boron hydrides. Diborane produced symptoms like metal fume fever and severe central nervous system irritation. Krachow (1953) reported that single inhalations of 50 ppm may be fatal to rats, resulting in lung injury, while 6 ppm repeatedly for three weeks causes lung damage, and 2 ppm causes some lung injury within four weeks. Kidney effects are also noted. He finds diborane about as injurious as phosgene. Odor is evident at 2 to 4 ppm.

The most important effects of diborane inhalation are central nervous system irritation and lung injury. The 0.1 ppm threshold limit can be interpreted from the effects of repeated animal inhalation and clinical studies on accidental human injuries. It is apparently low enough to prevent injury.

O-Dichlorobenzene. Cameron, Thomas, Ashmore, Warren, Buchan, and Kenny-Hughes (1937) found 30 minutes inhalation of 390 ppm caused in animals, liver necrosis and minor kidney injury. They concluded it is more toxic than chlorobenzene. Fairhall (1949, p. 284) points out its narcotic properties. Elkins (1950, p. 147) reports some irritation of eye and respiratory tract from 100 ppm, without other effects.

The most important effect of o-dichlorobenzene inhalation is chronic poisoning centering in the liver. The 50 ppm threshold limit can be interpreted from the results of single animal inhalations and human sensory response data. It does not appear to allow sufficient margin to prevent human injury from continuous inhalation.

Dichlorodifluoromethane. Sayers, Yant, Chornyak and Shoaf (1930) found animals exposed repeatedly to 200,000 ppm developed a generalized tremor and ataxic gait, but no gross pathology. Fairhall (1949, p. 347) notes it has little, if any anesthetic or toxic action.

The most important effect of dichlorodifluoromethane inhalation is asphyxia from extremely high concentrations. The 1000 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It represents good engineering control rather than a hazard limit.

1,1-Dichloroethane. Henderson and Haggard (1943, p. 207) conclude it is similar to carbon tetrachloride. Smyth (1937-55) found rats survive eight hours at 4000 ppm, but are killed at 16,000 ppm, an acute toxicity half that of carbon tetrachloride. In repeated inhalations by rats and dogs, chronic toxicity somewhat less than that of carbon tetrachloride was likewise found.

The most important effect of 1,1-dichloroethane inhalation is chronic poisoning, centering in the liver. The 100 ppm threshold limit can be interpreted from single and repeated animal inhalations. It may be low enough to prevent injury, but new data are desirable in view of current views on carbon tetrachloride.

1,2-Dichloroethylene. Fairhall (1949, p. 292) concludes 39,000 to 50,000 ppm is lethal to guinea pigs, and 18,000 ppm produces narcosis. Acute poisoning consists of narcosis with central nervous system irritation. No liver injury has been found. The vapors are irritating. Smyth (1937-55) found the cis isomer did not kill nor anesthetize rats in four hours at 8000 ppm, while 16,000 ppm anesthetized in eight minutes and killed in four hours. The trans isomer was twice as toxic and anesthetic.

The most important effect of 1,2-dichloroethylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of single animal inhalations. It is low enough to prevent definite narcosis.

Dichloroethyl ether. Schrenk, Patty and Yant (1933) found 500 to 1000 ppm killed guinea pigs in 30 to 60 minutes with lung hemorrhage and edema, while 35 ppm produced slight irritation in several hours. This concentration can be smelled but is not immediately irritating to man, while 500 to 1000 ppm is lacrimating. Smyth (1937-55) found rats survive four hours at 125 ppm, but are killed by 250 ppm. Skin penetration is moderately dangerous.

The most important effect of dichloroethyl ether inhalation is lung injury. The 15 ppm threshold limit can be interpreted from the results of single animal inhalations. It

seems to be low enough to prevent injury.

Dichloromonofluoromethane. Nuckolls (1933) showed it is little different from other fluorocarbons used as refrigerants. They are practically inert in the body.

The most important effect of dichloromonofluoromethane inhalation is asphyxia from extremely high concentrations. The 1000 ppm threshold limit can be interpreted by analogy with other fluorocarbon refrigerants. It represents good engineering control rather than a hazard limit.

1,1-Dichloro-1-nitroethane. Machle, Scott, Treon, Heyroth and Kitzmiller (1945) found 25 ppm did not kill animals in a total of 204 hours inhalation. The vapors irritated eyes, nose, bronchi and lungs, with injury to liver, kidney and vascular system.

The most important effect of 1,1-dichloro-1-nitroethane inhalation is lung injury. The 10 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Dichlorotetrafluoroethane. Nuckolls (1933) and Yant, Schrenk and Patty (1932) found only transient discomfort in animals exposed two hours to 25,000 ppm. No chronic effects are to be expected from this physiologically inert material.

The most important effect of dichlorotetrafluorethane inhalation is asphyxia from very high concentrations. The 1000 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears to be far below a possible injurious level.

Dieldrin. The workers examined by Princi and Spurbeck (1951) after three years industrial exposure to several related insecticides including dieldrin with concentrations of the order of 5 mg./cu.m., showed no evidence of clinical effect. Treon and Cleveland (1955) found 25 ppm in the diet of rats for two years did not shorten their lives. Dogs are most sensitive, tolerating only 3 ppm. Chronic toxicity centered in the liver. In acute poisoning, central nervous system irritation is prominent. Dieldrin penetrates the skin.

The most important effect of dieldrin inhalation is chronic poisoning centering in the liver. The 0.25 mg./cu.m. threshold limit can be interpreted from the results of repeated oral doses to animals and results of examination of exposed workmen. It is low enough to prevent injury.

Diethylamine. Brieger and Hodes (1951) exposed rabbits repeatedly to 50 ppm and found lung and corneal injury, but animals survived with some liver injury. Smyth (1937-55) found rats suffer fractional mortality from four hours at 2000 and 4000 ppm, while 8000 is lethal to all. The liquid is extremely injurious to the cornea.

The most important effect of diethylamine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 25 ppm threshold limit can be interpreted from results of repeated animal inhalation. It appears low enough to prevent injury.

Difluorodibromomethane. ACGIH (1955b) quotes Chemical Corps Medical Laboratories Research Report No. 180, 1953. This shows six weeks daily inhalation of 2300 ppm kills some animals with lung injury, liver and central nervous system damage.

The most important effects of difluorodibromomethane inhalation are chronic toxicity and respiratory tract irritation. The 100 ppm threshold limit can be interpreted from the results of single animal inhalations, in comparison with carbon tetrachloride, ethyl and methyl bromide. It appears low enough to prevent injury.

Diisobutyl ketone. Silverman, Schulte and First (1946) found concentrations above 25 ppm give eye irritation in unacclimated subjects. Carpenter, Pozzani and Weil (1953) found single eight-hour inhalations of 200 ppm killed some rats by anesthesia, repeated inhalation of 1650 ppm killed some and caused liver, kidney and lung injuries, while 125 ppm was without effect. Two humans found 50 ppm satisfactory for work during a three-hour period, but were uncomfortable at 100 ppm.

The most important effect of diisobutyl ketone inhalation is narcosis. The 50 ppm threshold limit can be interpreted from repeated animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

2,4-Diisocyanotoluene. Swenson, Holmquist and Lundgren (1955) quote French animal inhalations in which 1 to 2 gm./cu.m. (140 to 280 ppm) caused only respiratory tract irritation. They report three human industrial cases featuring upper respiratory tract irritation followed by sensitization

and asthma-like attacks. Unpublished American experience features sensitization.

The most important effect of 2,4-diisocyanotoluene inhalation is respiratory tract irritation, followed by sensitization. The 0.1 ppm tentative threshold limit cannot be interpreted quantitatively. It is probably not low enough to prevent an attack in a sensitized person.

Dimethyl aniline (N-dimethyl aniline). Henderson and Haggard (1943, p. 227) conclude that the alkyl anilines are less toxic than aniline, but von Oettingen (1941, p. 15) concludes dimethyl aniline has a greater depressant effect than aniline. It forms methemoglobin in the blood. He cites two human poisonings with symptoms like aniline.

The most important effect of dimethyl aniline inhalation is poisoning like that from aniline. The 5 ppm threshold limit can be interpreted by analogy with aniline. It appears low enough to prevent injury.

Dimethyl sulfate. Flury and Zernik (1931) report that 13 ppm severely poisoned cats in 20 minutes. Patty (1948-9, p. 925) states it has only a faint odor and there is a considerable latent period before effects are evident. Fairhall (1949, p. 309) concludes it is a powerful irritant upon inhalation, the liquid causes severe skin burns and corneal injury, and when swallowed, marked central nervous system effects such as convulsions and delirium result. Smyth (1937-55) found rats survive four hours inhalation of 15 ppm but die from 30 ppm.

The most important effect of dimethyl sulfate inhalation is delayed irritation of bronchi, and lung edema, not preceded by promptly evident irritation of eye and upper respiratory tract. Very high concentrations may cause convulsions and delirium, then coma. The 1 ppm threshold limit can be interpreted from results of single animal inhalations. It appears to be low enough to protect against lung injury, but available data do not indicate that it will prevent bronchial irritation.

Dinitrobenzene. Fairhall (1949) concludes the chief effect of dinitrobenzene is the production of methemoglobin, leading to anoxia and anemia. Von Oettingen (1941) in a review of the literature finds chronic liver injury and cites opinions that it is more toxic than nitrobenzene.

The most important effect of inhalation of dinitrobenzene is chronic poisoning. The 1 mg./cu.m. tentative threshold limit appears to be based on the reasonable assumption that dinitrobenzene is five times as toxic as nitrobenzene.

Dinitro-o-cresol. Baltimore (1943) reports a non-fatal case from inhalation of 4.7 mg./cu.m. Spencer, Rowe, Adams and Irish (1948) in animal experiments, found 10 to 50 mg./kg. is a fatal dose for animals. It is a rapidly acting metabolic stimulant, increasing body temperature to the point of heat stroke. Cataracts are produced in susceptible species, but chronicity is low.

The most important effect of dinitro-o-cresol inhalation is acute poisoning, marked by metabolic stimulation with rise of body temperature. The 0.2 mg./cu.m. threshold limit can be interpreted from the facts of one industrial accident. It appears low enough to prevent injury.

Dinitrotoluene. Von Oettingen (1941, p. 110) concludes this is similar to trinitrotoluene but less toxic when pure. The dust causes mucous membrane irritation.

The most important effect of dinitrotoluene inhalation is chronic poisoning, marked by central nervous system, liver and red blood cell changes. The 1.5 mg./cu.m. threshold limit can be interpreted from analogy with trinitrotoluene. It does not appear low enough to prevent all injuries.

Dioxane. Fairley, Linton and Ford-Moore (1934) found liver and kidney injury in animals repeatedly inhaling 1000 ppm, and from skin absorption. Silverman, Schulte and First (1946) found eye, nose and throat irritation at 300 ppm in unacclimated subjects. Patty (1948-9, p. 957) concludes there is only a faint odor at 200 ppm. Smyth (1937-55) found rabbits particularly susceptible, repeated inhalation at 800 ppm killing some with kidney injury within 30 days.

The most important effect of dioxane inhalation is chronic poisoning, centering in the liver and kidney. The 100 ppm threshold limit can be interpreted from results of repeated animal exposure studies. It appears to be low enough to prevent injury.

EPN. Hodge, Maynard et al (1954) found the acute oral LD_{50} for rats ranges from 7 to 33 mg./kg., while 75 ppm in the diet is without effect during two years. The ma-

terial is a cholinesterase inhibitor, and symptoms of excess in animals are confined to excitability and tremors. It appears to be ⅛ to ⅓ as toxic as parathion.

The most important effect of EPN inhalation is the reduction of blood cholinesterase. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of single oral doses to animals and analogy with parathion. It appears low enough to prevent injury.

Ethyl acetate. The unacclimated subjects of Nelson, Ege, Ross, Woodman and Silverman (1943) found an objectionably strong odor at 200 ppm and eye, nose and throat irritation at 400 ppm. Henderson and Haggard (1943, p. 222) conclude 10,000 to 20,000 ppm is dangerous for short exposures. It is mildly narcotic but does not produce systemic effects. Smyth (1937-55) found rats inhaling 8000 ppm for four hours survive, but 16,000 ppm kills.

The most important effect of ethyl acetate inhalation is narcosis. The 400 ppm threshold limit can be interpreted by human sensory data and results of single inhalations by animals. It apparently is low enough to prevent definite narcosis.

Ethyl acrylate. Pozzani, Weil and Carpenter (1949) found 30 inhalations of 300 ppm injured lungs, liver and kidneys of rats and rabbits, while 70 ppm did not injure rats. They found that 8 ppm is readily detectable by odor, and that the odor of 50 ppm is objectionable. Treon, Sigmon, Wright and Kitzmiller (1949) obtained closely similar results.

The most important effect of ethyl acrylate inhalation is respiratory tract irritation. The 25 ppm tentative threshold limit can be interpreted from repeated animal inhalation results and limited human sensory data. It appears low enough to prevent injury.

Ethyl alcohol. Henderson and Haggard (1943, p. 219) consider its vapors anesthetic but not toxic. Only under exceptional circumstances can inhalation cause slight intoxication. They estimate 1064 ppm for eight hours is not sufficient to produce the earliest stage of intoxication in man. Cook (1945) stated that industrial exposure at 1000 ppm has led only to a few complaints of eye irritation. Patty (1948-9, p. 851) concludes 6000 to 9000 ppm is initially intolerably irritating, but that acclimatization follows. Smyth (1937-55) found rats survive eight hours at 16,000 ppm but some are killed at 32,000 ppm.

The most important effect of ethyl alcohol inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from human physiological data. It is low enough to prevent significant narcosis, but some eye irritation will result.

Ethyl amine. Brieger and Hodes (1951) exposed rabbits repeatedly to 50 ppm and found lung and corneal injury, with some effect on heart muscle. Smyth (1937-55) found four hours at 4000 to 8000 ppm kills some rats, and 16,000 ppm kills all. Respiratory tract irritation is prominent. The liquid is extremely injurious to the cornea.

The most important effect of ethyl amine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 25 ppm threshold limit can be interpreted from results of repeated animal inhalation. It appears low enough to prevent injury.

Ethyl benzene. Yant, Schrenk, Waite and Patty (1930) found only slight irritation in guinea pigs breathing 1000 ppm for eight hours. Fatalities from higher levels showed lung edema. Humans found 1000 ppm was initially irritating to the eyes and 2000 ppm caused throat irritation, constriction in the chest and slight intoxication. Smyth (1937-55) found rats not killed by four hours at 2000 ppm, while 8000 ppm is fatal. There is general agreement that it does not exert the hematopoetic effects of benzene.

The most important effect of ethyl benzene inhalation is narcosis, with irritation of the entire respiratory tract contributing to injury. The 200 ppm threshold limit can be interpreted from results of single animal inhalations and limited human sensory response. It is low enough to prevent injury.

Ethyl bromide. Waite and Yant (1928) found lung edema a prominent effect of guinea pig inhalation. Sayers, Yant, Thomas and Berger (1929) found several hours inhalation of 1700 ppm by animals was without effect. Henderson and Haggard (1943, p. 207) note that vapors are not narcotic.

The most important effect of ethyl bromide inhalation is respiratory tract irritation. The 200 ppm threshold limit can be interpreted from the results of single animal inhalations. It is probably low enough to prevent injury.

Ethyl chloride. Sayers, Yant, Thomas and Berger (1929) found with animals that 150,000 to 300,000 ppm is rapidly fatal, death being due to anesthesia, that 40,000 ppm does not kill in 270 minutes, 20,000 ppm causes only moderate unsteadiness, and 10,000 ppm is without effect.

The most important effect of ethyl chloride inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from the results of single animal inhalations. It is low enough to prevent significant narcosis.

Ethylene chlorhydrin. Dierker and Brown (1944) reported one fatality with estimated exposure of 305 ppm for two hours. Animal experiment produced kidney pathology from 365 ppm for two hours. Goldblatt (1944) and Goldblatt and Chiesman (1944) report 11 cases, two of them fatal. Nervous system, cardiovascular system and kidneys were affected, and no warning irritation was apparent. They found one hour at 1120 ppm killed animals, with evidence of potential chronic effect. They conclude no concentration is safe for daily exposure. Smyth and Carpenter (1945) point out very rapid skin penetration of the liquid, the absence of warning skin irritation and the failure of rubber gloves to protect.

The most important effect of ethylene chlorhydrin inhalation is acute poisoning, centering in the kidney. The 5 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears low enough to prevent injury.

Ethylene diamine. Dernehl (1951) related industrial experience showing this is a sensitizer upon contact and inhalation. Pozzani and Carpenter (1954) exposed rats repeatedly to the vapors. All died within 20 days at 484 ppm, with loss of hair, injury to the kidney and lesser effects on liver and lung. There was no effect except loss of hair at 132 ppm, and none whatever at 59 ppm. Very brief human exposures found 100 ppm inoffensive, tingling of skin and nose at 200 ppm and intolerable sensory response at 400 ppm. Smyth (1937-55) found eight hours at 2000 ppm did not kill rats while 4000 ppm was fatal. Death was due chiefly to kidney injury, with some injury to lung. The liquid irritates the skin and severely injures the cornea.

The most important effects of ethylene diamine inhalation are respiratory tract irritation, kidney damage and sensitization. The 10 ppm threshold limit can be interpreted from results of repeated animal inhalation and human sensory data. It is low enough to prevent irritation and injury, but probably not to eliminate response by persons already sensitized.

Ethylene dibromide. Rowe, Spencer, McCollister, Hollingsworth and Adams (1952) found four species of animals tolerated repeated inhalation of 25 ppm, but not 50 ppm. Major injury was in lung and liver, with kidney and central nervous system less prominent. The liquid penetrates the skin. It is painful in the eye, but causes only transient injury. The odor of a concentration dangerous to life is definite and sickening.

The most important effects of ethylene dibromide inhalation are respiratory tract irritation and liver injury. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It is probably low enough to prevent injury.

Ethylene dichloride. Spencer, Rowe, Adams, McCollister and Irish (1951) studying repeated inhalation by animals, found no effect from 100 ppm. Single dangerous inhalations irritate the lung and depress the central nervous system, while dangerous repeated inhalations injure liver and kidney. They feel chronic intoxication is unlikely because tolerated repeated inhalations are close to concentrations tolerated once. Concentrations sufficient to cause marked narcosis are irritating to the upper respiratory tract. Adams, Spencer, Rowe, McCollister and Irish (1952) simultaneously studying carbon tetrachloride, found it at least four times as toxic as ethylene dichloride. Elkins (1950, p. 137) found complaints of nausea from industrial exposures to 100 to 150 ppm. Patty (1948-9, p. 805) finds little odor at 100 ppm, slight eye and nose irritation at 1000 ppm.

The most important effect of ethylene dichloride inhalation is chronic poisoning, centering in the liver. The 100 ppm threshold limit can be interpreted from the results of repeated animal inhalations and human response. It is low enough to prevent injury.

Ethylene imine. Silver and McGrath (1948) and Carpenter, Smyth and Shaffer (1948) studied single inhalations in animals. The LC_{50} for mice in a 10-minute exposure

is 2236 ppm, 500 ppm for one hour is fatal to rats and guinea pigs, 25 ppm kills some in eight hours, and 10 ppm kills none in eight hours. Symptoms and death are delayed, due to kidney tubular injury, with lesser lung and liver injury. Humans can barely smell 2 ppm, while 100 ppm begins to irritate eyes and nose. The liquid penetrates the skin, produces severe skin and corneal burns, and sensitizes the skin.

The most important effect of ethylene imine inhalation is acute poisoning, centering in the kidney, with lung injury of lesser importance. The 5 ppm threshold limit can be interpreted from results of single animal inhalation studies and limited human sensory data. It is apparently low enough to prevent poisoning and upper respiratory tract irritation.

Ethylene oxide. Waite, Patty and Yant (1930) in single animal inhalations found no symptoms from eight hours at 250 ppm. Greater concentrations caused eye and nose irritation, narcosis, bronchial and lung irritation. Sensory response is only moderate at low concentrations, but eye and nose irritation are intolerable at high concentrations. Sexton and Henson (1950) have called attention to spectacular human skin injuries with sensitization, which arise from contact with the liquid and its aqueous solutions. Smyth (1937-55) found rats survive four hours at 4000 ppm but are killed by 8000 ppm. Hollingsworth, Rowe, Oyen, McCollister and Spencer (1956) in animals repeatedly inhaling 204 ppm found lung irritation and some fatalities, with injury to liver, kidney, adrenal and testes. Rats and mice were not affected at 49 ppm, other species tolerated 113 ppm. Jacobson, Hackley and Feinsilver (1956) with dogs, rats and mice found some fatalities from repeated inhalation of 400 ppm, but 100 ppm had little affect.

The most important effect of ethylene oxide inhalation is respiratory tract irritation leading to lung injury, while injury to liver and kidney are less important. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. It appears low enough to prevent injury.

Ethyl ether. Henderson and Haggard (1943, p. 195) estimate the maximum human blood concentration from inhalation of 400 ppm is 0.018 gm./l., causing no intoxication, while 2000 ppm will give a blood level of 0.09 gm./l., corresponding to instability in some persons. They state 35,000 ppm anesthetizes in 30 minutes, and a higher concentration kills by respiratory paralysis. Experience in human anesthesia shows that pneumonitis may follow ether anesthesia, but other injuries are unlikely. Nelson, Ege, Ross, Woodman and Silverman (1943) found nasal irritation at 200 ppm with unacclimated subjects, somewhat greater at 300 ppm.

The most important effect of ethyl ether inhalation is narcosis. The 400 ppm threshold limit can be interpreted from human physiological and sensory data. It is low enough to prevent definite narcosis.

Ethyl formate. Flury and Zernik (1931) reported 330 ppm causes in man slight eye irritation and rapidly increasing nasal irritation, while 10,000 ppm is anesthetic and fatal. Fairhall (1949, p. 344) notes its effects are irritation and narcosis, and that there is no chronic toxicity. Smyth (1937-55) found rats survive four hours inhalation of 4000 ppm but die from 8000 ppm.

The most important effect of ethyl formate inhalation is narcosis. The 100 ppm threshold limit can be interpreted from scanty human sensory data and single animal inhalations. It appears to be low enough to prevent definite narcosis and irritation.

Ethyl mercaptan. Sayers, Fieldner, Yant, Leitch and Pearce (1930) report the odor detectable at one part per billion and disagreeable at one part per fifteen million. They quote that its effects are like those of hydrogen sulfide, and by analogy with butyl mercaptan they conclude more than 733 ppm is required to kill in 30 minutes. Flury and Zernik (1931) quote 3000 ppm as harmless to dogs and 10,000 ppm causing hematologic and blood cell changes.

The most important effect of ethyl mercaptan is eye and respiratory tract irritation. The 250 ppm tentative threshold limit can be interpreted from the results of single inhalation by animals. It appears low enough to prevent injury and eye irritation.

Ethyl silicate. Smyth and Seaton (1940) found eight-hour inhalation of 550 ppm the least fatal exposure for guinea pigs. Death is due to lung injury, with some kidney

damage. Humans found 85 ppm detectable by odor, 250 ppm slightly irritating to eye and nose, and 3000 ppm extremely irritating. Rowe, Spencer and Bass (1948) found some kidney damage in rats repeatedly inhaling 125 ppm. Pozzani and Carpenter (1951) exposed rodents repeatedly. Some died within 30 days at 440 ppm with injury to lung, liver and kidney, but 88 ppm did not injure. The data suggest that repeated inhalation at a given concentration is no more injurious than a single inhalation.

The most important effect of ethyl silicate inhalation is lung injury, with non-progressive kidney damage less important. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations and limited human sensory data. It can be smelled, it is not irritating, and is low enough to prevent lung or kidney injury.

Ferbam. Hodge, Maynard, Downs, Blanchet and Jones (1952) reported the oral LD_{50} for rats to be over 17 gm./kg., with guinea pigs and rabbits more sensitive. Rats tolerated 0.01% in their diet for 30 days without effect while 0.5% was required to kill. Dogs were not injured by 25 mg./kg./day for six months. The mechanism of injury is not clear.

The most important effect of inhalation of Ferbam appears to be the upper respiratory tract irritation of a substantially inert dust. The 15 mg./cu.m. tentative threshold limit is in accord with this.

Ferro vanadium dust. Roshchin (1952) exposed rats two months to 1000 to 2000 mg./cu.m. and found no effect beyond some lung irritation. The author suggests a threshold limit of 1 mg./cu.m. Vanadium compounds irritate the upper respiratory tract, but Sjoberg (1951) found no chronic general poisoning in workers exposed to vanadium pentoxide dust, symptoms being confined to respiratory difficulties and skin allergies.

The most important effect of inhalation of ferrovanadium dust is respiratory tract irritation. The 1 mg./cu.m. threshold limit can be interpreted from limited repeated animal inhalations and examination of exposed workmen. It appears low enough to prevent injury.

Fluoride dust. Roholm (1937) found fluorosis of human bone, but no other effects, after several years work in 2 to 3 ppm fluorine, equivalent to 1.5 to 2.3 mg./cu.m. soluble fluoride dust. Higher concentrations provide respiratory tract irritation and effects on liver and kidney. Largent (1952) found storage in the body occurs when as little as three milligrams per day fluoride in the form of sodium fluoride is ingested, roughly equivalent to inhalation of 0.3 mg./cu.m. soluble fluoride dust.

The most important effect of inhalation of fluoride dust is chronic poisoning, centering in the bones, with respiratory tract irritation at high concentrations. The 2.5 mg./cu.m. threshold limit can be interpreted from results of examination of exposed workmen and experimental studies of human fluoride retention. It is not low enough to prevent fluoride storage with resulting effects on the bones.

Fluorine. Machle and Evans (1940) examined workmen exposed intermittently to as much as 10 ppm and found no clinical evidence of damage, but there was some accumulation in bones and teeth. Stokinger (1949) found few toxic effects in dogs repeatedly exposed to 0.5 ppm. Greater concentrations injured lung and kidney. This is apparently below the level which leads to bone abnormalities (Roholm 1937).

The most important effect of inhalation of fluorine gas is respiratory tract irritation, with lung edema the maximum effect. Chronic effect on bone metabolism is also important. The 0.1 ppm threshold limit can be interpreted from results of animal inhalation and studies on exposed workmen. It is low enough to prevent injury.

Fluoroacetates. Dieke and Richter (1946) report the median lethal oral dose to be 0.22 mg./kg. for wild rats. The substance is rapidly fatal through intervention in the tricarboxylic acid metabolic cycle.

The most important effect of fluoroacetate inhalation is acute toxicity. The 0.1 mg./cu.m. tentative threshold limit can be interpreted from acute oral toxicity data for rats. It corresponds to a maximum human intake of one milligram per day, apparently well below a dangerous amount.

Fluorotrichloromethane. Nuckolls (1933) found in animals no more than occasional tremors and retching during two hours at 22,000 to 25,000 ppm. No toxic effects are to be expected from this physiologically inert material.

The most important effect of fluorotrichloromethane inhalation is a minor degree of narcosis, and asphyxia from very high concentrations. The 1000 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears to be far below a possibly injurious level.

Formaldehyde. Henderson and Haggard (1943, p. 128) conclude its action is chiefly irritation of all tissues contacted, particularly the respiratory tract, and that systemic effects are not important. Skin sensitization to solutions is frequent and respiratory tract sensitization to the gas is not unlikely. Elkins (1950, p. 231) reports irritation in workmen inhaling 5 to 6 ppm, and eye irritation of unhardened persons at lower levels. Smyth (1937-55) found rats survive eight hours inhalation of 125 ppm but are killed by 250 ppm. The liquid causes severe corneal injury.

The most important effect of formaldehyde inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 5 ppm threshold limit can be interpreted from uncontrolled human sensory data. It is sufficiently low to prevent lung injury.

Furfural. Fairhall (1949, p. 354) quotes animal experiments in which inhalation of 280 ppm resulted only in slight mucous membrane irritation, while 2800 ppm caused acute irritation, prostration and lung edema. ACGIH (1954b) quotes Korenman and Resnik, (*Arch. Hyg.*, 104:344, 1931) to the effect that 2 to 14 ppm causes human headache and eye irritation. Severe corneal injury is to be expected from the fluid, and analogy with other aldehydes suggests that skin and respiratory sensitization may be found.

The most important effect of furfural inhalation seems to be irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 5 ppm tentative threshold limit can be interpreted from uncontrolled human sensory data. It is sufficiently low to prevent lung injury.

Furfuryl alcohol. Fine and Wills (1950) found that death from furfuryl alcohol is due to the respiratory paralysis of anesthesia, and that short of death, its effects are reversible. ACGIH (1955b) quotes Chemical Corps Medical Laboratories Research Report No. 139, 1942, to the effect that eight hours inhalation of 700 ppm killed 25% of a group of rats.

The most important effect of furfuryl alcohol inhalation is narcosis. The 50 ppm tentative threshold limit can be interpreted from results of single animal inhalations. It appears low enough to prevent injury.

Gasoline. Sayers, Fieldner, Yant and Thomas (1927) reported human dizziness at 700 ppm, and Drinker, Yaglou and Warren (1943) found human respiratory tract irritation and headache begin at 1000 ppm. Elkins (1950, p. 99) found industrially no sensory response to 660 to 800 ppm benzine, and dizziness at 2000 to 3000 ppm. He concludes chronic effects do not occur when concentrations are too low to cause narcosis, but Hayhurst (1936) reported chronic poisoning consisting of central nervous system effects and blood cell changes after many years of exposure. This article does not seem to be generally accepted. Henderson and Haggard (1943, p. 192) consider nausea and incoordination the significant effects, with anesthetic death at 20,000 to 30,000 ppm. Aromatic hydrocarbons in gasoline from cracking operations may much reduce safety.

The most important effect of gasoline inhalation is narcosis. The threshold limit of 500 ppm can be interpreted from human sensory data, both experimental and industrially observed. It is low enough to prevent definite narcosis.

Heptane. Patty and Yant (1929) reported slight human dizziness from 1000 ppm. The paraffin hydrocarbons are anesthetic agents and irritate mucous membrane, but they do not cause systemic toxicity.

The most important effect of heptane inhalation is narcosis. The 500 ppm threshold limit can be interpreted from limited human sensory data and by analogy with the better studied gasoline. It is probably low enough to prevent definite narcosis.

HETP (hexaethyltetraphosphate). This material is substantially identical in quantitative and qualitative effect with the cholinesterase inhibitor TEPP. Apparently no data specifically upon inhalation have been published.

The most important effect of HETP is reduction of blood cholinesterase. The 0.1 mg./cu.m. tentative threshold limit can be interpreted by analogy with parathion. It

appears to be consistent with that value.

Hexane. Nelson, Ege, Ross, Woodman and Silverman (1943) with unacclimated subjects found no irritation at 500 ppm. Drinker, Yaglou and Warren (1943) found human nausea, headache, eye and throat irritation at 1400 to 1500 ppm. The paraffin hydrocarbons anesthetize and irritate mucous membrane, but do not cause systemic toxicity.

The most important effect of hexane inhalation is narcosis. The 500 ppm threshold limit can be interpreted from human sensory data. It is low enough to prevent definite narcosis.

Hexanone (methyl butyl ketone). Simple ketones are irritant and narcotic, but not chronically toxic. Schrenk, Yant and Patty (1936) found guinea pigs tolerate 1000 ppm with slight or no symptoms for several hours, a concentration with a strong odor, moderately irritating to human eyes and nose. Death from higher levels is anesthetic respiratory paralysis. Specht, Miller, Valaer and Sayers (1940) found it more depressant than acetone, butanone or pentanone. Smyth (1937-55) found rats survive four hours at 4000 ppm, but die at 8000 ppm.

The most important effect of hexanone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from the results of single animal inhalations and limited human response data. It is low enough to prevent definite narcosis.

Hexone (methyl isobutyl ketone). Simple ketones are irritant and narcotic, but not chronically toxic. Specht (1938) and Specht, Miller, Valaer and Sayers (1940) found guinea pigs are not injured by several hours at 1000 ppm, but men find eye and nose irritation. Smyth (1937-55) found rats survive four hours at 2000 ppm, but die from 4000 ppm. Silverman, Schulte and First (1946) found the odor objectionable at 200 ppm, eye irritation evident, but no nose and throat irritation with unacclimated subjects.

The most important effect of hexone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from results of single animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

Hydrazine. Comstock, Lawson, Greene and Oberst (1954) found in animals damage to lung and liver, tremors and convulsions, irritation of eye, nose and throat. Most of the animals repeatedly inhaling 20 ppm died by 30th day. During six month's inhalation of 5 ppm, only minor changes in the lungs of rats and dogs were found.

The most important effect of inhalation of hydrazine is respiratory tract irritation. The 1 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is probably low enough to prevent injury.

Hydrogen Bromide. ACGIH (1955b) quotes unpublished human response data from the Connecticut Bureau of Industrial Hygiene. Odor was evident at 2 ppm. nose and throat irritation began to be evident at 3 ppm, and eye irritation was not evident at 6 ppm.

The most important effect of hydrogen bromide inhalation is respiratory tract irritation, with lung edema the maximum effect. The 5 ppm tentative threshold limit can be interpreted from analogy with hydrogen chloride and human sensory response. It is probably low enough to prevent injury.

Hydrogen chloride. Machle, Kitzmiller, Scott and Treon (1942) found animals not affected by repeated inhalation of 34 ppm. Henderson and Haggard (1943, p. 126) consider it an irritant without systemic effect. They state 1000 to 2000 ppm is dangerous to life through lung edema in a short time. Elkins (1950, p. 79) finds 10 ppm highly irritating to humans although immunity seems to develop; 5 ppm immediately irritating; and even lower levels can erode the teeth.

The most important effect of hydrogen chloride inhalation is respiratory tract irritation, with lung edema the maximum effect. The 5 ppm threshold limit can be interpreted from repeated animal inhalation and human sensory data. It is low enough to prevent injury.

Hydrogen cyanide. Flury and Zernik (1931) give 19 to 36 ppm as tolerable for six hours without symptoms. Henderson and Haggard (1943, p. 173) conclude injury is chemical asphyxia, and chronic toxicity is not to be expected. They state 3000 ppm is rapidly fatal, 100 to 240 ppm dangerous in 30 to 60 minutes, and 20 to 40 ppm gives slight symptoms in several hours. This is one of the few gases which can penetrate the skin in dangerous amounts. Patty (1948-9, p. 633) finds the odor barely detectable at 0.9 ppm, and recognizable at 2 to 5 ppm.

The most important effect of hydrogen cyanide inhalation is acute poisoning, a chemical asphyxia. The 10 ppm threshold limit can be interpreted from results of older single animal inhalation data and some human sensory data. It is low enough to prevent injury.

Hydrogen fluoride. Stokinger (1949) found 30 ppm is highly toxic to animals, causing pulmonary damage, kidney and testis changes and increases in bone fluoride. Animals tolerated 7 ppm with only mild respiratory tract irritation in repeated exposure. Elkins (1950, p. 73) reports nosebleeds at 0.4 to 0.7 ppm. Patty (1948-9, p. 543) finds 0.026 mg./l. (22 ppm) is slowly irritating and at 0.1 mg./l. (120 ppm) the skin smarts. The liquid causes severe slowly healing skin injuries, and destroys the cornea. All soluble fluorides interfere with calcium metabolism and an excess produces bone and tooth abnormalities.

The most important effect of hydrogen fluoride inhalation is respiratory tract irritation, with lung edema the maximum effect. Chronic effect on bone metabolism is also important. The 3 ppm threshold limit can be interpreted from results of repeated animal inhalation and human sensory data. It is low enough to prevent injury

Hydrogen peroxide, 90%. Oberst, Comstock and Hackley (1954) found rats survive eight hours at 250 to 300 ppm without symptoms, but irritation and areas of edema are found in the lungs. Dogs survived six months at 7 ppm without injury, although the skin was thickened and the lungs irritated. The liquid is extremely destructive to skin and cornea.

The most important effect of hydrogen peroxide aerosol inhalation is respiratory tract irritation, with lung edema the maximum effect. The 1 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent injury.

Hydrogen selenide. Dudley and Miller (1941) found animals are killed in eight hours at 0.3 to 1.2 ppm, with lung irritation and changes in liver and spleen. Eye and nose irritation made 1.5 ppm intolerable to man, but 0.3 ppm is not irritating and perception of its odor is soon lost. Buchan (1947) reported industrial cases due to less than 0.2 ppm, with liver injury.

The most important effect of hydrogen selenide is acute poisoning, largely lung edema at high concentration, and chronic poisoning centering in the liver. The 0.05 ppm threshold limit can be interpreted from the results of single animal inhalations and industrial accidents. It appears low enough to prevent injury

Hydrogen sulfide. Henderson and Haggard (1943, p. 140, 243) state hydrogen sulfide may cause very rapid death from respiratory paralysis, or delayed death from lung injury. It is not cumulative. Low concentrations irritate the cornea. A concentration fatal in 30 minutes is 600 ppm, while 70 to 150 ppm causes slight symptoms in several hours. Barthelemy (1939) found no injury among viscose workers during 10 years, with control at about 20 ppm. Elkins (1950, p. 232) found eye irritation in industry at 10 ppm and some complaints even at 5 ppm. Patty (1948-9, p. 590) concludes 0.3 ppm can be smelled, 3 to 5 ppm is offensive.

The most important effect of hydrogen sulfide inhalation is acute toxicity, marked by respiratory paralysis or lung edema. The 20 ppm threshold limit can be interpreted from the results of examination of exposed workmen. It is low enough to prevent injury.

Hydroquinone. Sterner, Oglesby and Anderson (1947) reported on several years industrial experience with men exposed to quinone vapor and hydroquinone dust. No systemic effects could be found, but high concentrations caused transient eye irritation, and after several years, a pigmentation of cornea and conjunctiva was apparent, due to local action on the exposed tissue. Loss of vision has followed pigmentation in some cases, according to Oglesby (1956). It is uncertain whether the vapor or the dust was responsible. Hydroquinone dust ranged from 0.12 to 13 mg./cu.m. After comparing exposure with concentration, the authors conclude hydroquinone dust should be kept below 2 to 3 mg./cu.m.

The most important effect of hydroquinone inhalation is transient eye irritation and a slowly developing pigmentation in the eye. Visual disability can result. The 2 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It appears low enough to prevent effect.

Iodine. Henderson and Haggard (1943, p. 133) state it is a respiratory tract irritant with more effect on the lungs than chlorine and bromine. They quote an 1889 thesis by Matt to the effect that 0.1 ppm does not disturb workers. Fairhall (1949, p. 90) points out that excessive absorption can disturb the metabolism through effect on the thyroid.

The most important effect of iodine vapor inhalation is respiratory tract irritation, with lung edema the maximum injury. The 0.1 ppm threshold limit can be interpreted from analogy with chlorine. It appears low enough to prevent injury.

Iron oxide fume. Fairhall (1949, p. 92) reviews several articles. Inhalation of iron oxide dust for 5 to 10 years can lead to a benign pneumoconiosis, siderosis, revealed by x-ray but not injurious. U.S. Department of Labor (1941) in studies of welders, concluded iron oxide fume below 30 mg./cu.m. was without effect, while in excess of this level a chronic bronchitis may result.

The most important effect of inhalation of iron oxide fume is bronchitis or metal fume fever. The 15 mg./cu.m. threshold limit can be interpreted by analogy with zinc oxide fume. It is low enough to prevent injury.

Isophorone. Smyth, Seaton and Fischer (1942) found no effect upon animals from repeated inhalation of 25 ppm, while 50 ppm caused some lung and kidney injury. Single inhalation of 4600 ppm (saturation) for eight hours injured the lung but did not anesthetize or kill. Silverman, Schulte and First (1946) found odor objectionable at 10 ppm and eye, nose and throat irritation at 25 ppm with unacclimated subjects.

The most important effects of isophorone inhalation are lung and kidney injury. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalation and human sensory data. It seems low enough to prevent injury.

Isopropylamine. Smyth, Carpenter and Weil (1951) reported that rats survive four hours inhalation of 4000 ppm, but die from 8000 ppm. This is half the acute toxicity Smyth (1937-55) found for butyl amine. ACGIH (1955b) cite unpublished industrial experience that levels above 5 ppm tend to be irritating.

The most important effect of isopropylamine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 5 ppm threshold limit can be interpreted from analogy with ethyl amines. It is probably low enough to prevent injury.

Lead. Russell, Jones, Bloomfield, Britten and Thompson (1933) after a survey of a storage battery plant, proposed that the threshold limit be reduced from 0.5 to 0.15 mg./cu.m. At this level disabling lead poisoning does not occur, and mild poisoning is rare.

The most important effect of lead dust inhalation is chronic poisoning. The 0.15 mg./cu.m. threshold limit can be interpreted from the results of examinations of exposed workmen. It is low enough to prevent disabling poisoning, but not to prevent some mild cases.

Lead arsenate. Fairhall and Miller (1941) and Fairhall, Miller and Weaver (1943) found the arsenate in lead arsenate decreases lead absorption or increases lead excretion, and reduces lead storage.

The most important effect of inhalation of lead arsenate is chronic arsenic poisoning. The 0.15 mg./cu.m. tentative threshold limit can be interpreted from the results of biochemical studies on animals. It appears to have been selected by analogy with the limit for lead, while analogy with arsenic would yield a much higher figure.

Lindane. A.M.A. (1951) in a summary, states lindane in large doses acts as a central nervous system stimulant, leading to hyperirritability, convulsions and death. Animals exposed several months to saturated vapors were not affected, but 10,000 mg./cu.m. dust for one hour killed one of two mice. ACGIH (1954b) quotes an unpublished 1951 report by J. F. Treon et al. A year of repeated inhalation of 0.7 mg./cu.m. caused minimal pathology in animals. An unpublished 1952 thesis by Spear is quoted to the effect that 655 days at 0.19 mg./cu.m. 24 hours a day did not result in pathology in rats.

The most important effect of lindane inhalation is chronic poisoning centering in the liver. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Magnesium oxide fume. Drinker, Thomson and Finn (1927) reported that experi-

mental fume fever in man results from excessive inhalation, but does not occur below a concentration of 15 mg./cu.m. This condition is transient fever with chills, muscular pain, nausea and vomiting. An immunity is apparently build up.

The most important effect of inhalation of magnesium oxide fume is transient metal fume fever. The 15 mg./cu.m. threshold limit can be interpreted from the results of extensive human experiment. It is low enough to prevent injury.

Malathon. Johnson, Fletcher, Nolan and Cassaday (1952) reviewed toxicity data and conclude malathon is about one-hundredth as toxic to mammals as parathion. Tousey (1954) reviews data and confirms that its toxicity is considerably lower than that of many other cholinesterase inhibitors. Culver, Caplan and Batchelor (1955) found a group of entomologists with maximum exposure about five hours at a peak of 56 mg./cu.m. and an average of about 3.3 mg./cu.m. This had no effect on blood cholinesterase.

The most important effect of malathon inhalation is the reduction of blood cholinesterase. The 15 mg./cu.m. threshold limit can be interpreted from the results of oral doses to animals and limited examinations of exposed workmen. It appears low enough to prevent injury.

Manganese. Flinn, Neal and Fulton (1951) describe poisoning as an effect upon the basal brain ganglia, leading to disability from weakness in the legs, spastic gait, stolid mask-like expression and emotional disturbances, but not ordinarily shortening life. In an ore-crushing plant, they found no symptoms in men exposed to 30 mg./cu.m. or less and they concluded concentrations can be effectively limited to 6 mg./cu.m.

The most important effect of inhalation of manganese dust is chronic poisoning. The 6 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It is low enough to prevent injury.

Mercury. Neal et al. (1941) in a study of the felt hat industry, found the incidence of mercurialism proportional to atmospheric concentrations, with no cases found below 0.1 mg./cu.m. Chronic symptoms consist of psychic disturbances, timidity, tremors, pallor, salivation and tenderness of the gums. Ashe, Largent, Dutra, Hubbard and Blackstone (1953) in repeated inhalations by animals, found no effects at 0.1 mg./cu.m., but damage to kidney and brain at 0.86 mg./cu.m.

The most important effect of inhalation of mercury is chronic poisoning. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalation and examination of exposed workmen.

Mercury (organic compounds). Ahlmark (1948) on the basis of Swedish industrial experience suggests a limit of 0.01 mg./cu.m. Lundgren and Swensson (1949) consider concentrations fluctuate so widely that analysis does not detect important peaks, and that an M.A.C. cannot be defined. Organo mercurials produce effects like mercury, but they have the added hazard of ready penetration of the skin. Trakhtenberg, (abstracted from the Russian in *Chemical Abstracts* 44:10162g, 1950) reported mice die at 10 to 30 mg./cu.m. within three to five hours and concludes 0.01 mg./cu.m. should not be tolerated for repeated human exposures.

The most important effect of inhalation of organo mercurials is chronic mercury poisoning. The 0.01 mg./cu.m. threshold limit can be interpreted from the results of industrial experience and single animal inhalations. It is probably low enough to prevent injury.

Mesityl oxide. Smyth, Seaton and Fischer (1942) found no effect upon animals from repeated inhalation of 50 ppm, while higher concentrations killed by anesthesia, with minor lung, kidney and liver injuries. In single inhalations, 100 ppm did not injure in eight hours, 500 killed some and 2,500 killed all, and in one hour 13,000 ppm (saturation) was fatal by anesthesia. Little cumulative action was revealed. Silverman, Schulte and First (1946) found some eye irritation at 25 ppm, and at 50 ppm nose irritation and a persistent unpleasant taste in unacclimated subjects.

The most important effect of mesityl oxide inhalation is narcosis. The 50 ppm threshold limit can be interpreted from the results of repeated animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

Methoxychlor. Haag, Finnegan, Larson, Riese and Dreyfuss (1950) found methoxy-

chlor acts similarly to DDT in animals, and is less toxic by inhalation. Hodge, Maynard and Blanchet (1952) found no effect on rats in two years at 0.020% in the diet and no mortality or histological changes at 0.16%. Little accumulates in body fat.

The most important effect of methoxychlor inhalation is chronic poisoning centering in the liver. The 15 mg./cu.m. threshold limit can be interpreted from the results of repeated oral doses to animals. It appears low enough to prevent injury.

Methyl acetate. Fairhall (1949, p. 375) notes irritation of eye and respiratory tract, narcosis less prominent than from higher acetates, but fatal dose close to anesthetic dose, and symptoms persistent after appreciable narcosis. Death is due to anesthesia, but lung injury also occurs. Smyth (1937-55) found rats survive four-hour inhalations of 16,000 ppm, but die from 32,000 ppm.

The most important effect of methyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted by analogy with ethyl acetate, allowing a margin for greater irritation and for the slow metabolism of methyl alcohol. It apparently is low enough to prevent narcotic symptoms.

Methyl Acetylene. ACGIH (1955b) cites Horn, Chemical Corps Medical Laboratories Contract Report #35, 1954. For six months dogs and rats inhaled 28,700 ppm repeatedly. A few died. There was lung irritation and some central nervous system excitation.

The most important effect of methyl acetylene inhalation is lung injury. The 1000 ppm threshold limit can be interpreted from results of repeated animal inhalations. It is low enough to prevent injury.

Methyl acrylate. Treon, Sigmon, Wright and Kitzmiller (1949) exposed animals repeatedly to vapor. They found that 130 seven-hour inhalations of 31 ppm had no effect upon four species, except some loss in weight. Higher concentrations caused respiratory tract irritation and some narcosis.

The most important effect of methyl acrylate inhalation is respiratory tract irritation. The 10 ppm tentative threshold limit can be interpreted from repeated animal inhalations. It appears to be low enough to prevent injury.

Methylal (dimethoxymethane). Weaver, Hough, Highman and Fairhall (1951) found that high concentrations produce fatty changes in liver, kidney and heart of animals, with lung irritation. The threshold for chronic effects is 11,300 ppm.

The most important effect of methylal inhalation is chronic poisoning, centering in liver and kidneys, with narcosis and lung injury less important. The 1000 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. It appears low enough to prevent injury, but there are no data to judge the degree of irritation and narcosis it allows.

Methyl alcohol. Sayers, Yant, Schrenk, Chornyak, Pearce, Patty and Linn (1942) found no effect on dogs from repeated inhalation of 450 to 500 ppm. Henderson and Haggard (1943, p. 218) stress slow elimination, leading to progressive rise in blood level from daily inhalation. At 200 ppm 0.87 grams can be absorbed by a human in eight hours, but only part of this can be eliminated before the next day. Methanol is primarily a narcotic agent, but it may injure retina and optic nerve, leading to cloudy vision or blindness. There is some irritation of mucous membranes. Elkins (1950, p. 111) found industrial exposures ranging from 100 to 1700 ppm with no evidence of poisoning. Smyth (1937-55) found rats survive eight-hour inhalations of 32,000 ppm, and only a fraction are killed by 64,000 ppm.

The most important effect of methyl alcohol inhalation is narcosis, with injury to retina and optic nerve likely only from quite excessive inhalation. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalations. It will not cause significant narcosis, but continuous inhalation will cause a daily rise in the degree of early narcosis, due to slow elimination.

Methyl bromide. Irish, Adams, Spencer and Rowe (1940) found no effect from repeated inhalation at 17 ppm, and 34 ppm injured only rabbits. Watrous (1942) found mild symptoms in one-third of 90 workers in concentrations generally under 35 ppm. Ingram (1951) found injuries where workers were exposed to 100 to 1000 ppm. After improvements reduced exposure to about 20 ppm, injuries ceased. Fairhall (1949, p. 376) notes it is a respiratory tract irritant, a liver injurant, and a central nervous system poison leading to delirium, convulsions and even mania. It is rapidly metabolized and eliminated.

The most important effect of methyl bromide inhalation is chronic poisoning, centering in the central nervous system and liver. The 20 ppm threshold limit can be interpreted from the results of repeated animal inhalations and examination of exposed workmen. It appears to be rather precisely set at the maximum concentration humans tolerate without effect.

Methyl CELLOSOLVE (methoxyethanol). Donley (1936) and Parsons and Parsons (1938) reported toxic encephalopathy with granulopenic anemia from industrial exposure to a mixed solvent containing methyl CELLOSOLVE. Greenberg, Mayers, Goldwater, Burke and Moskowitz (1938) estimated concentrations in the establishment some time after the cases developed, and found 25 ppm of the glycol ether. Werner, Mitchell, Miller and von Oettingen (1943, a,b) in experiments on dogs did not confirm the degree of toxicity suggested by the human cases. The blood cell effects were obtained from repeated inhalation of 500 ppm, but no encephalopathy was found. Fairhall (1949, p. 336) concludes the vapors are somewhat irritating, and produce narcosis and kidney changes. Smyth (1937-55) found rats survive four hours at 2000 ppm, but die from eight hours.

The most important effect of methyl CELLOSOLVE inhalation is chronic poisoning, centering in the brain and red blood cells. The 25 ppm threshold limit can be interpreted from atmospheric analyses of doubtful validity after human industrial injuries, and from the results of repeated animal inhalations. It appears lower than is required to prevent injuries.

Methyl CELLOSOLVE acetate (2-methoxyethyl acetate.) This ester hydrolyzes in the body to methyl CELLOSOLVE and acetic acid, and its vapors are somewhat more irritating than those of the former.

The most important effect of methyl CELLOSOLVE acetate inhalation is chronic poisoning due to hydrolysis to methyl CELLOSOLVE. The 25 ppm threshold limit can be interpreted from analogy with methyl CELLOSOLVE. It is low enough to prevent injury.

Methyl chloride. Sayers, Yant, Thomas and Berger (1929) found 10 hours at 400 ppm produce no serious injuries in guinea pigs. Smith and von Oettingen (1947) found repeated inhalation of 300 ppm by six species has no effect, but 500 ppm causes central nervous system effects. Complete recovery from injury is slow. Fairhall (1949, p. 379) concludes it acts chiefly by narcosis, but liver, kidney and bone marrow injuries are found.

The most important effect of methyl chloride inhalation is central nervous system injury, with less important chronic poisoning. The 100 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Methyl chloroform. Adams, Spencer, Rowe and Irish (1950) found in repeated exposures that 650 ppm retarded guinea pig growth, and 3000 ppm caused slight liver effects. Three other species were less sensitive. Rats were mildly narcosed in one hour at 5000 ppm. The level tolerated in repeated inhalation was close to that tolerated in a single exposure. They concluded it is close to methylene chloride in toxicity and less toxic than trichloroethylene.

The most important effect of methyl chloroform inhalation is narcosis. The 500 ppm threshold limit can be interpreted from the results of repeated animal inhalation. It is low enough to prevent definite narcosis.

Methyl cyclohexane. Treon, Crutchfield and Kitzmiller (1943) found no effect on rabbits exposed 300 hours to 1162 ppm, slight kidney and liver changes from 90 hours at 2886 ppm and fractional mortality, eye and entire respiratory tract irritation and narcosis at 7308 ppm. Patty (1948-9, p. 770) concludes the odor is weak at 500-800 ppm.

The most important effect of methylcyclohexane inhalation is narcosis, with nonprogressive organic changes. The 500 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent definite narcosis.

Methyl cyclohexanol. Treon, Crutchfield and Kitzmiller (1943) found 300 hours at 121 ppm causes slight liver and kidney changes in rabbits, and 300 hours at 503 ppm causes eye irritation with some narcosis, but does not kill. Patty (1948-9, p. 881) concludes that odor and irritation are evident at 500 ppm.

The most important effect of methyl cyclohexanol inhalation is narcosis, with non-progressive organic effects less promi-

nent. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent significant narcosis, and probably to prevent minor effects on liver and kidneys.

Methyl cyclohexanone. Treon, Crutchfield and Kitzmiller (1943) found no effect on rabbits from 300 hours at 182 ppm, slight eye irritation at 514 ppm, while 1822 ppm caused some narcosis and eye irritation.

The most important effect of methyl cyclohexanone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations. It appears low enough to prevent definite narcosis.

Methylene chloride. Heppel, Neal, Perrin, Orr and Porterfield (1944) found repeated inhalation of 10,000 ppm caused moderate narcosis in animals, some deaths from lung edema and liver damage. A concentration of 5000 ppm for six months had no effect upon four species, but reduced the voluntary activity of rats, indicative of a very early stage of narcosis not usually detected in animals. Fairhall (1949, p. 296) mentions a human fatality after accidental anesthesia.

The most important effect of inhalation of methylene chloride is chronic poisoning centering in the liver. The 500 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Methyl formate. Schrenk, Yant, Chornyak and Patty (1936) found guinea pigs tolerate 1500 to 2000 ppm for several hours without disturbance, and 5000 ppm for one hour. Symptoms of higher concentrations were nose and eye irritation, lung irritation, narcosis and anesthetic death.

The most important effect of methyl formate inhalation is narcosis. The 100 ppm threshold limit can be interpreted from results of single inhalations by animals. It appears to be low enough to prevent definite narcosis.

Methyl isobutyl carbinol (methyl amyl alcohol). Silverman, Schulte and First (1946) at 50 ppm found eye irritation in unacclimated subjects, although the odor was not objectionable. Experience with other alcohols indicates systemic effects are not to be expected. Smyth (1937-55) found rats inhaling 1000 ppm for eight hours are not killed, but 2000 ppm is fatal.

The most important effect of methyl isobutyl carbinol inhalation is narcosis. The 25 ppm threshold limit can be interpreted from human sensory data. It is low enough to prevent significant narcosis and irritation.

Methyl mercaptan. ACGIH (1954b) quotes de Rikowski who in 1893 found methyl mercaptan similar to but somewhat less toxic than hydrogen sulfide.

The most important effect of methyl mercaptan is eye and respiratory tract irritation. The 50 ppm tentative threshold limit can be interpreted by analogy with hydrogen sulfide. It appears low enough to prevent injury and eye irritation.

Molybdenum. Fairhall, Dunn, Sharpless and Pritchard (1945) in animal experiments, found some bronchial and alveolar irritation, with fatty changes in liver and kidneys. Animals survived repeated exposure one hour daily to 53 mg./cu.m. molybdic oxide fume and only one of a group was killed by 286 mg./cu.m. molybdenite dust. The Industrial Hygiene Digest (16:1083, 1952) abstracts Mogilevskaya to the effect that histopathological changes in rat heart, liver and kidney are found after repeated inhalation of 3 to 10 mg./cu.m. molybdenum oxide aerosol.

The most important effect of inhalation of molybdenum compounds is chronic poisoning, centering in liver and kidney. The threshold limits of 5 mg./cu.m. for soluble and 15 mg./cu.m. for insoluble molybdenum compounds can be interpreted from the results of repeated animal inhalations. They appear low enough to prevent injury, but slight toxic effects may result from soluble compounds.

Naphtha (coal tar). A mixture of toluene and xylene chiefly. The predominantly narcotic effects of these closely similar materials are additive and a mixture is no more injurious than the sum of its components. Cook (1945) points out that if a sample has a low boiling point, an appreciable content of benzene is to be suspected, and working concentrations should be reduced accordingly.

Being composed chiefly of toluene and xylene, the most important effect of coal tar naphtha inhalation, when free from benzene, is narcosis with irritation of the respiratory tract less important. The 200 ppm

threshold limit can be interpreted by analogy with those of toluene and xylene. It is low enough to prevent injury.

Naphtha (petroleum). A mixture of paraffin hydrocarbons of somewhat higher molecular weight than gasoline. The discussion under gasoline applies.

Nickel carbonyl. Fairhall (1949, p. 114) quotes Armit to the effect that this produces a deposit of finely divided nickel in the respiratory tract, leading to irritation and lung edema. Hueper (1950) summarizes the inferences that it produces cancer, originating in the nasal sinuses. Kincaid, Strong and Sunderman (1953) found 30 minutes at 10 ppm killed mice, 270 ppm killed cats, and they suggest toxicity may be related to body weight, with man surviving high concentrations. They found lung edema and severe liver injury, but conclude it is not cumulative and that a tolerance develops. Sunderman and Kincaid (1954) report on 36 human cases, two fatal. The fatalities were delayed, due to lung edema.

The immediate effect of nickel carbonyl vapor inhalation is lung irritation and delayed edema. Cancer originating in the nasal sinuses has been reported from longtime exposure. The 0.001 ppm threshold limit is apparently an approximation of zero, designed to prevent cancer. It is low enough to prevent all possibility of immediate effect, but there are no data to judge its effectiveness in preventing cancer.

Nicotine. Wilson and De Eds (1936) fed diets containing nicotine to growing rats for a 60-day period. Rats did not survive on 0.05% nicotine. Rats were not affected by 0.006% nicotine, equivalent to 4 mg./kg. body weight per day. A greater concentration reduced growth, due largely but not entirely to reduced food intake. Lehman (1949) estimates the fatal human dose to be 60 milligrams.

The most important effect of nicotine inhalation is ill-defined chronic poisoning. The 0.5 mg./cu.m. tentative threshold limit can be interpreted from repeated feeding studies on rats. It corresponds to a maximum human intake of five milligrams per day, apparently well below an injurious level.

Nitric acid. Fairhall (1949, p. 81) concludes it is an upper respiratory tract irritant, injuring the bronchi and even the lungs in high concentration. It also erodes the teeth. He suggests a threshold limit of 10 ppm.

The most important effect of inhalation of nitric acid is upper respiratory tract irritation. The 10 ppm tentative threshold limit appears to be based upon undocumented analogy with other acid gases.

p-Nitroaniline. Fairhall (1949, p. 402) concludes p-nitroaniline is more toxic than aniline, causing headache, nausea and cyanosis. This is based on British industrial experience.

The most important effect of p-nitroaniline inhalation is acute poisoning. The 1 ppm threshold limit is apparently an estimate based on analogy with aniline. It appears low enough to prevent injury.

Nitrobenzene. Henderson and Haggard (1943, p. 227) note that injury by skin absorption is more frequent than by inhalation. The compound is an anesthetic, producing methemoglobin and reducing blood pressure. Acute toxicity is marked by headache, narcosis, cyanosis, and death from respiratory paralysis. Chronic absorption results in anemia, cyanosis, muscular weakness, bladder irritation. The maximum concentration which gives no serious disturbance in one hour is 200 ppm, and 40 to 80 ppm cause symptoms in several hours.

The most important effect of nitrobenzene inhalation is chronic poisoning, marked by reduced blood pressure and cyanosis. The 1 ppm threshold limit can be interpreted from results of single animal inhalation. It appears low enough to prevent injury.

Nitroethane. Machle, Scott and Treon (1940) found guinea pigs inhaling 500 ppm for a total of 140 hours are not injured, but some die from 1000 ppm. Eye and nose irritation, narcosis, central nervous system irritation and lung edema are produced. Toxic symptoms are evident before narcosis.

The most important effect of nitroethane inhalation is acute poisoning accompanied by narcosis and irritation. The 100 ppm threshold limit can be interpreted from results of animal inhalation. It appears low enough to prevent injury.

Nitrogen dioxide. Henderson and Haggard (1943, p. 137) state that 62 ppm causes immediate throat irritation, 300 ppm coughing and 100 to 150 ppm is danger-

ous for 30 to 60 minutes. Fairhall (1949, p. 117) finds the vapor irritates the entire respiratory tract, leading to delayed lung edema. There is some reduction in blood pressure, causing headache. Gray, McNamee and Goldberg (1952) found respiratory tract inflammation in rats, inhaling 9 ppm for a total of 48 hours in 10 days. Patty (1948-9, p. 610) reports 5 ppm is evident by odor, 10 to 20 ppm is irritating to eyes and nose. Vigliani and Zurlo (1955) in workers exposed several years to 30 to 35 ppm found no symptoms. They regard a threshold limit of 15 ppm satisfactory when ozone is absent.

The most important effect of nitrogen dioxide inhalation is respiratory tract irritation, with delayed lung edema probable. The 5 ppm threshold limit can be interpreted from results of repeated animal inhalation and human sensory data. It appears low enough to prevent injury.

Nitroglycerine. Cook (1945) quotes U. S. Public Health Service experience of no systemic effects from 10 ppm, but with as little as 0.5 ppm causing severe headache upon return to work after a week-end. Fairhall (1949, p. 405) notes headache from lowered blood pressure, excitement, dizziness, fainting, cyanosis, death from respiratory paralysis. Acclimatization is prominent, skin penetration is a major hazard. Elkins (1950, p. 160) found some headaches at 0.04 ppm.

The most important effect of nitroglycerine inhalation is acute poisoning, marked by reduced blood pressure. The 0.5 ppm threshold limit can be interpreted from studies on exposed workmen. It is sufficiently low to prevent injury, but not to prevent headache.

Nitromethane. Machle, Scott and Treon (1940) found animals not affected by 500 ppm for a total of 140 hours, but 1000 ppm was fatal. The vapors are eye and respiratory irritants and mildly narcotic. Central nervous system irritation and lung edema result. Toxic symptoms are evident before narcosis.

The most important effect of nitromethane inhalation is acute poisoning, accompanied by narcosis and irritation. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalation. It appears low enough to prevent injury.

2-Nitropropane. The animal work of Machle, Scott and Treon (1940) concluded that in the nitro paraffins toxicity increases with molecular weight. Thus, 2-nitropropane should be more toxic than nitroethane. Skinner (1947) found workmen in concentrations of 10 to 30 ppm were not affected, but 20 to 45 ppm caused anorexia, nausea, vomiting and diarrhea.

The most important effect of 2-nitropropane inhalation is acute poisoning accompanied by narcosis and irritation. The 50 ppm threshold limit can be interpreted from results of repeated animal inhalation and industrial experience. It is probably low enough to prevent injury, but not to prevent disturbing symptoms.

Nitrotoluene. Von Oettingen (1941) reviewed the literature and could find no clear distinction between the toxicities of nitrotoluene and nitrobenzene.

The most important effect of nitrotoluene inhalation is chronic poisoning, marked by reduced blood pressure and cyanosis. The 5 ppm threshold limit appears to be an estimate without quantitative support. It is doubtful whether the difference between nitrobenzene and nitrotoluene is sufficient to justify the difference in threshold limits.

Octane. No useful published data specifically upon octane were found, but analogy with heptane and gasoline is close.

The most important effect of octane inhalation is narcosis. The 500 ppm threshold limit can be interpreted only by analogy with pentane and gasoline. It is probably low enough to prevent definite narcosis.

Ozone. Fairhall (1949, p. 122) quotes McDonnell's incompletely reported work to the effect that daily inhalation of 0.1 ppm killed guinea pigs with pneumonia, higher concentrations leading to lung edema. He notes respiratory tract irritation with fatal pneumonitis or lung edema, but no systemic poisoning. He quotes a statement that 0.015 ppm can be smelled, and any higher concentration is irritating.

The most important effect of ozone inhalation is respiratory tract irritation, with lung edema the maximum effect. The 0.1 ppm threshold limit can be interpreted from results of limited repeated animal inhalations and human sensory data. It appears low enough to prevent injury.

Parathion. The earliest effect of this cholinesterase inhibitor is a reduction of

that enzyme activity in the blood. Brown and Bush (1950) in tests on industrial workers found inhalation of 0.1 to 0.8 mg./cu.m. causes definite reduction in blood cholinesterase activity. Vigliani and Zurlo (1955) on the basis of cholinesterase reduction conclude that a threshold limit of 0.07 to 0.12 mg./cu.m. is satisfactory.

The most important effect of parathion inhalation is the reduction of blood cholinesterase. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It is not certain that it is low enough to prevent measurable reduction of blood cholinesterase, but it is low enough to prevent injury.

Pentaborane. Svirbely (1954a,b) found the LC_{50} for mice inhaling vapors for two hours to be 10.9 ppm. Symptoms were central nervous excitability and corneal opacity. Six-hour inhalations of 3.3 ppm by rats killed all within four repetitions. Comstock and Oberst (1953) report that the median detectable odor is 2.5 mg./cu.m. (0.5 ppm), described as garlic or slightly sweet.

The most important effect of pentaborane inhalation is acute toxicity involving the central nervous system. The 0.01 ppm tentative threshold limit can be interpreted from limited repeated inhalations by animals. It appears to be a conservative estimate.

Pentachlornaphthalene. After extensive inhalation studies with rats, Drinker (1939) concluded that injury is exclusively in the liver and that the permissible limit for repeated inhalation is 0.5 mg./cu.m. The solid material or its oil solutions penetrates the skin, and repeated contact leads to chloracne.

The most important effect of pentachlornaphthalene inhalation is chronic poisoning centering in the liver. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Pentachlorophenol. Kehoe, Deichmann-Gruebler and Kitzmiller (1939) found no evidence of chronic poisoning in rabbits, and the smallest lethal intravenous dose was 22 mg./kg. It penetrates the skin readily. Internal injury is primarily to the vascular system with heart failure.

The most important effect of pentachlorophenol inhalation is acute poisoning centering in the circulatory system. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated doses to animals. It appears low enough to prevent injury.

Pentane. Patty and Yant (1929) found no effect on humans from 10 minutes inhalation of 5000 ppm. Fairhall (1949, p. 358) concludes that only narcosis and irritation are produced.

The most important effect of pentane inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from limited human sensory data and analogy with the better studied gasoline. It is probably low enough to prevent definite narcosis.

Pentanone (methyl propyl ketone). Yant, Patty and Schrenk (1936) found 30,000 to 50,000 ppm killed guinea pigs in 30 to 60 minutes; 1500 ppm caused slight or no symptoms in several hours and was strongly odorous and irritating to human eye and nose. Death was anesthetic. Smyth (1937-55) found four hours at 2000 ppm killed part of a group of rats.

The most important effect of pentanone inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of single animal inhalation and limited human response data. It is low enough to prevent definite narcosis.

Perchloroethylene. Carpenter (1937) found rats inhaling 230 ppm for 150 days showed slight non-progressive liver and kidney effects, while 70 ppm had no effect. Humans perceived the odor at 50 ppm, slight eye irritation at 500 ppm, light narcosis at 1000 ppm, nausea at 5000 ppm. Rowe, McCollister, Spencer, Adams and Irish (1952) found repeated inhalation of 400 ppm does not affect rats, rabbits and monkeys, while 100 ppm does not affect guinea pigs. Humans found no symptoms at 100 ppm, minimum narcosis at 200 ppm, eye and nose irritation at 600 ppm, painful irritation at 1000 ppm.

The most important effect of perchloroethylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of repeated animal inhalations and human response. It appears low enough to prevent significant narcosis.

Perchloromethyl mercaptan. ACGIH (1954b) cites Flury and Zernik (1931) to the effect that after 15 minutes inhalation of

45 ppm, mice and cats die within two days.

The most important effect of perchloromethyl mercaptan inhalation is eye and respiratory tract irritation. The 0.1 ppm tentative threshold limit can be interpreted on the basis of limited single inhalations by animals. It appears to be low enough to prevent injury.

Phenol. Deichmann, Kitzmiller and Witherup (1944) found guinea pigs are severely injured by 20 days inhalation of 25 to 50 ppm, rabbits suffer lung injury in 63 days, but rats are not affected. They conclude human injury from repeated inhalation is marked by digestive disturbance, nervous disorders, skin eruption and liver and kidney damage. The liquid penetrates the skin to a dangerous extent, and causes severe skin and corneal injury. Patty (1948-9, p. 1034) reports that 5 ppm can be recognized by odor. Smyth (1937-55) found rats survive eight hours inhalation of vapors saturated at room temperature.

Although phenol vapors are odorous and irritating, their major effect is chronic systemic poisoning. The 5 ppm threshold limit can be interpreted from results of repeated inhalations by animals. It appears to be low enough to prevent chronic toxic effects.

Phenylhydrazine. Von Oettingen (1941, p. 158) concludes death from a large dose is due to respiratory paralysis. There is hemolytic anemia, formation of methemoglobin, injury to the liver and heart muscle. Skin penetration is rapid and dermal sensitization takes place. The fatal oral dose for rats is of the order of 0.04 gm./kg.

The most important effect of phenylhydrazine inhalation is chronic poisoning, centering in the red blood cells. The 5 ppm threshold limit can be interpreted from analogy with aniline. Quantitative data are not available to judge the effectiveness of the limit.

Phosgene. Fieldner, Katz and Kinne (1921) cite the Chemical Warfare Service as authority for a 1 ppm allowable concentration for prolonged exposure, based on human tests. Henderson and Haggard (1943, p. 137) consider phosgene a lung injurant producing delayed edema. They say 3.1 ppm is immediately irritating to throat, 4 ppm to eyes, 4.9 ppm causes coughing, 5.6 ppm detectable by odor and 50 ppm rapidly fatal.

The most important effect of phosgene inhalation is respiratory tract irritation with delayed lung edema probable. The 1 ppm threshold limit can be interpreted from results of human studies. It is low enough to prevent injury.

Phosphine. Henderson and Haggard (1943, p. 243) report 1000 to 2000 ppm fatal in 30 minutes, 100 to 200 ppm the maximum for one hour without serious disturbance. Fairhall (1949, p. 127) quotes Müller to the effect that animals die from two four-hour inhalations of 20 ppm, or 7 of 10 ppm, but two months at 5 ppm did not injure. Acute poisoning is rapidly fatal with convulsions, paralysis and coma. Chronic poisoning is marked by anemia and nervous disturbances. Patty (1948-9, p. 576) concludes there is only a faint odor at 1 ppm.

The most important effect of phosphine inhalation is chronic poisoning. The 0.05 ppm threshold limit can be interpreted from the results of repeated animal inhalation. It appears low enough to prevent injury.

Phosphorous (yellow). Fairhall (1949, p. 131) summarizes the literature. The effects of chronic poisoning are upon bone metabolism. They may be noticed first as painful swollen gums, or as spontaneous fractures of the long bones. They develop into periostitis and necrosis of the lower jaw, with secondary infection. In single exposures one milligram per kilogram is usually fatal.

The most important effect of phosphorous inhalation is chronic poisoning, centering in the bones. The 0.1 mg./cu.m. threshold limit cannot be interpreted in quantitative terms.

Phosphorous pentachloride. Henderson and Haggard (1943, p. 134) conclude this material is an irritant to nose, throat and lungs through hydrolysis to hydrogen chloride. Skin burns from the solid are likely. Mice are killed in 10 minutes by 120 ppm.

The most important effect of phosphorous pentachloride inhalation is respiratory tract irritation, with lung edema possible. The 1 mg./cu.m. threshold limit is apparently an estimate without quantitative support. It appears low enough to prevent injury.

Phosphorous pentasulfide. Fairhall (1949, p. 131) quotes Barillet to the effect that phosphorous pentasulfide is somewhat less hazardous than phosphorous pentachloride.

The most important effect of phosphorous pentasulfide inhalation is respiratory tract

irritation. The 1 mg./cu.m. threshold limit is apparently an estimate without quantitative support. It appears low enough to prevent injury.

Phosphorous trichloride. Henderson and Haggard (1943, p. 134) consider it an irritant and lung injurant. They cite 600 ppm as rapidly fatal, and 2 to 4 ppm as the maximum for 30 to 60 minutes without serious disturbance. Cook (1945) quotes Butjog as finding 0.7 ppm causes only slight irritation in animals.

The most important effect of phosphorous trichloride inhalation is respiratory tract irritation, with lung edema the maximum effect. The 0.5 ppm threshold limit can be interpreted from limited animal inhalation data. It appears low enough to prevent injury.

Picric acid. Fairhall (1949, p. 423) describes systemic poisoning as gastroenteritis, hemorrhagic nephritis and hepatitis. Sunderman, Weidman and Batson (1945) studied workers handling ammonium picrate in atmospheres from 0.0088 to 0.1942 mg./cu.m. They found little respiratory tract irritation, no systemic effects but considerable dermatitis.

The most important effect of picric acid inhalation is chronic poisoning. The 0.1 mg./cu.m. threshold limit can be interpreted from observations on exposed workmen. It is low enough to prevent systemic injury but not respiratory tract irritation and sensitization.

Propyl acetate. Fairhall (1949, p. 426) concludes it is more irritating than ethyl acetate, more narcotic than ethyl or methyl acetates, but less lethal. Some respiratory tract irritation and liver injury are found. Death is due to anesthesia, but even deep narcosis may leave no after effects. Smyth (1937-55) found four hours inhalation of 32,000 ppm kills four of six rats.

The most important effect of propyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted by analogy with ethyl acetate, not by data. It appears to be low enough to prevent definite narcosis.

Propyl alcohol, iso. Nelson, Ege, Ross, Woodman and Silverman (1943) found 400 ppm causes mild irritation of eye, nose and throat, and 800 ppm is no more severe in unacclimated subjects. Fairhall (1949, p. 429) concludes it is similar to ethyl alcohol with no delayed effects, but twice as toxic. Smyth (1937-55) found rats survive four hours at 12,000 ppm, but half are killed in eight hours.

The most important effect of isopropyl alcohol inhalation is narcosis. The 400 ppm threshold limit can be interpreted from human sensory data and analogy with ethyl alcohol. It is low enough to prevent significant narcosis, but some irritation will result.

Propylene dichloride. Heppel, Neal, Highman and Porterfield (1946) found repeated inhalation of 1000 ppm by animals killed the first animal in seven days with severe liver effects. They conclude it is more toxic than ethylene dichloride but less so than carbon tetrachloride.

The most important effect of propylene dichloride inhalation is chronic poisoning centering in the liver. The 75 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Propylene imine. Carpenter, Smyth and Shaffer (1948) found rats and guinea pigs killed by four hours at 500 ppm, and not by 30 minutes. This is about $\frac{1}{8}$th the acute toxicity of ethylene imine in simultaneous work.

The most important effect of propylene imine vapor inhalation is acute poisoning, centering in the kidney, with lung injury of lesser importance. The 25 ppm threshold limit can be interpreted from results of scanty single animal inhalation studies and analogy with ethylene imine. It is apparently low enough to prevent injury.

Propyl ether (isopropyl ether). Machle, Scott and Treon (1939) found incomplete anesthesia in animals at 30,000 ppm, light narcosis at 10,000 ppm, and no effect in repeated exposures at 1000 ppm. They conclude it is 1.5 to 2 times as active as ethyl ether and less so than gasoline.

The most important effect of isopropyl ether inhalation is narcosis. The 500 ppm threshold limit can be interpreted from results of repeated animal inhalation. In comparison with data on ethyl ether it does not seem low enough to prevent definite narcosis.

Pyrethrum. There is little published on the toxicity of pyrethrum but many years of wide use as an insecticide indicates a low

degree of hazard, except for some slight skin sensitizing property. Carpenter, Weil, Pozzani and Smyth (1950) found the rat oral LD_{50} of two samples to be 0.82 and 1.87 gm./kg. Inhalation studies were made with an insecticidal aerosol containing 10% pyrethrum and 90% peanut oil and a Freon propellant. Rats inhaled a concentration of 6000 mg./cu.m. pyrethrum with 10 times as much peanut oil for 30 minutes, and only moderate lung congestion resulted. Rats and dogs inhaled a concentration of 16 mg./cu.m. pyrethrum with 10 times as much peanut oil for 40 thirty-minute periods during 31 calendar days, without injuries greater than those in peanut oil controls. Lehman (1949) estimates the fatal human dose to be 100 grams.

The most important effect of pyrethrum inhalation is irritation of the upper respiratory tract. The 2 mg./cu.m. tentative threshold limit can be interpreted from limited repeated animal inhalation data. It appears low enough to prevent injury.

Pyridine. Pollock, Finkelman and Arieff (1943) using pyridine for human therapy, found no toxic symptoms after daily doses of 0.31 to 1.54 ml., but 1.85 to 2.46 ml. was toxic, with one death of liver and kidney injury. Fairhall (1949, p. 434) considers small repeated doses affect the bone marrow, increasing the platelet count. Elkins (1950, p. 167) quotes a Czech report of mild central nervous symptoms at 6 to 12 ppm.

The most important effect of pyridine inhalation is chronic poisoning, centering in liver, kidney and bone marrow. The 10 ppm threshold limit can be interpreted from limited human symptom data. Mild symptoms may be found.

Quinone. Sterner, Oglesby and Anderson (1947) reported on several years industrial experience with men exposed to quinone vapor and hydroquinone dust. No systemic effects could be found, but high concentration caused transient eye irritation, and after several years a pigmentation of cornea and conjunctiva was apparent, due to local action on the exposed tissue. Loss of vision has followed pigmentation in some cases, according to Oglesby (1956). It is uncertain whether the vapor or the dust was responsible. Concentrations from 0.01 to 3.2 ppm quinone were found in the plant. The odor of quinone is perceptible at about 0.1 ppm, definite at about 0.15 ppm, irritating at 0.5 ppm and markedly irritating at 3 ppm. After comparing exposure with concentration, the authors conclude quinone vapor should be kept below 0.1 ppm.

The most important effect of quinone inhalation is transient eye irritation and a slowly developing pigmentation in the eye. The 0.1 ppm threshold limit can be interpreted from the results of examination of exposed workmen. It appears low enough to prevent effect.

Rotenone. On the basis of the literature and his own work, Lehman (1949) estimates the fatal human dose to be 200 grams by mouth.

The most important effect of rotenone inhalation is irritation of the upper respiratory tract. The 5 mg./cu.m. tentative threshold limit can be interpreted by analogy with pyrethrum. It appears low enough to prevent injury.

Selenium compounds (as Se). Fairhall (1949, p. 145) summarizes experimental studies which stress oral doses of selenium dioxide and inhalation of hydrogen selenide. Even at 3 ppm dioxide in the diet rats are injured, while 10 ppm kills within eight weeks. Dudley and Miller (1941) found animals killed in eight hours at 0.3 to 1.2 ppm hydrogen selenide, primarily due to lung injury, with changes in liver and spleen. Buchan (1947) reports industrial cases due to less than 0.2 ppm (0.65 mg./cu.m.) hydrogen selenide, with symptoms largely referable to the liver.

The most important effect of inhalation of selenium-bearing dusts is chronic poisoning, centering in the liver. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of repeated feeding to animals and by analogy with hydrogen selenide. This is probably low enough to prevent injury.

Sodium hydroxide. Elkins (1950, p. 84) states inhalation of mists result in upper respiratory tract irritation leading to ulceration. Patty (1949, p. 561) on the basis of experience with caustic mists from 1 to 40 mg./cu.m., concludes a concentration of 2 mg./cu.m. is noticeably but not excessively irritating.

The most important effect of sodium hydroxide mist or dust inhalation is upper respiratory tract irritation, leading to ulceration. The 2 mg./cu.m. threshold limit

can be interpreted from observations upon exposed workmen. It appears low enough to prevent injury.

Stibine. Webster (1946) finds stibine a lung injurant and powerful hemolytic agent, injuring liver and kidney as well. Some animals die after one hour inhalation of 40 ppm. Its action resembles that of the better understood arsine.

The most important effect of stibine inhalation is acute poisoning, largely lung edema. The 0.1 ppm threshold limit can be interpreted by analogy with arsine. It appears low enough to prevent injury.

Stoddard Solvent. Nelson, Ege, Ross, Woodman and Silverman (1943) found 400 ppm produced no marked effects on unacclimated subjects. This solvent is toxicologically identical with gasoline and remarks under that material apply.

Strychnine. McNally (1937) reports that a human death has resulted from swallowing 30 milligrams. It is a convulsive poison.

The most important effect of strychnine inhalation is acute poisoning. The 0.15 mg./cu.m. tentative threshold limit can be interpreted from the known human fatal dose. It corresponds to a maximum intake of 1.5 milligrams in a working day, apparently low enough to prevent injury.

Styrene monomer. Spencer, Irish, Adams and Rowe (1942) found immediate animal death was anesthetic, delayed death was due to lung injury. Blood cells were not affected. Repeated inhalation of 650 ppm had no effect on animals. Humans found 1300 ppm extremely irritating to eye and nose, and 400 ppm had an objectionable odor but little irritation. Carpenter, Shaffer, Weil and Smyth (1944) found irritation and early narcosis in humans at 800 ppm.

The most important effect of styrene vapor inhalation is narcosis. The threshold limit of 200 ppm can be interpreted from results of repeated animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

Sulfur dioxide. Kehoe, Machle, Kitzmiller and LeBlanc (1932) studied many workmen continuously exposed to sulfur dioxide and found only upper respiratory tract chronic irritation. Henderson and Haggard (1943, p. 131) conclude it is an irritant without systemic effect. They give 400 to 500 ppm as dangerous in a short time, 20 ppm irritating the eye and causing coughing, 8 to 12 ppm irritating the throat, and 3 to 5 ppm detectable by odor. Elkins (1950, p. 81) finds slight human irritation at 2 ppm and objectionable irritation at 10 to 30 ppm.

The most important effect of sulfur dioxide inhalation is respiratory tract irritation, with lung edema or respiratory arrest the maximum effect. The 10 ppm threshold limit can be interpreted from human sensory data and examination of exposed workmen. It is low enough to prevent injury.

Sulfur hexafluoride. Lester and Greenberg (1950) found that it is a physiologically inert gas, rats inhaling 800,000 ppm in oxygen for 16 to 24 hours were not affected.

The most important effect of sulfur hexafluoride inhalation is asphyxia from very high concentrations. The 1000 ppm threshold limit can be interpreted from the results of prolonged animal inhalations. It is so far below any possible injurious level that it represents good engineering control, rather than a hazard limit.

Sulfuric acid. Fairhall (1949, p. 83) considers it an upper respiratory tract irritant with lung injury possible. It is corrosive to the skin and eyes, and erodes the teeth. Amdur, Silverman and Drinker (1952) with normal human subjects, found changes in respiration at a concentration as low as 0.35 mg./cu.m., and pronounced decrease in minute volume at 5 mg./cu.m. Elkins (1950, p. 82) considers concentrations above 1 ppm (4 mg./cu.m.) are irritating. Sterner (1943) considered 5 mg./cu.m. as tolerable.

The most important effect of sulfuric acid inhalation is respiratory tract irritation, with lung edema possible. The 1 mg./cu.m. threshold limit can be interpreted from human sensory and physiological data. It is low enough to prevent injury.

Sulfur monochloride. Henderson and Haggard (1943, p. 130) consider it an upper respiratory tract irritant through release of hydrochloric acid, but rarely a lung injurant. Mice die from one minute inhalation of 150 ppm, cats from 15 minutes at 48 ppm. Fairhall (1949, p. 160) concludes chronic systemic effects do not occur. Elkins (1950, p. 81) found 2 to 9 ppm mildly irritating to humans.

The most important effect of sulfur monochloride vapor inhalation is respiratory

tract irritation. The 1 ppm threshold limit can be interpreted from results of single animal inhalations and human sensory data. It is low enough to prevent injury.

Sulfur pentafluoride. Greenberg and Lester (1950) found this to be a lung injurant. Rat lungs were severely injured by one hour at 10 ppm, less severely injured at 1 ppm and not affected at 0.1 ppm. Sixteen hours at 1 ppm was lethal, due to lung injury, while 18 hours at 0.5 ppm injured lungs but did not kill.

The most important effect of sulfur pentafluoride vapor inhalation is lung injury. The 0.025 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is probably low enough to prevent injury.

TEDP. This cholinesterase inhibitor is about half as toxic in single doses to animals as parathion, and the class of cholinesterase inhibiting compounds manifests little tendency to chronic effect.

The most important effect of TEDP inhalation is the reduction of blood cholinesterase. The 0.2 mg./cu.m. threshold limit can be interpreted only by analogy with parathion. It appears low enough to prevent injury.

Tellurium. Steinberg, Massari, Miner and Rink (1942) examined workmen exposed to tellurium fume ranging from 0.01 to 0.1 mg./cu.m., with a peak of 0.74 mg./cu.m. in one sample. The symptoms found were garlic odor of breath and sweat, dryness of mouth, metallic taste and somnolence. No signs of poisoning were found. Excessive absorption would have been shown by gastro-intestinal disturbances, reduction in red blood cells, diminished reflexes, and tremor.

The most important effect of inhalation of tellurium-containing dusts is chronic poisoning centering in the liver. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It appears low enough to prevent injury.

TEPP. This cholinesterase inhibitor is about twice as toxic in single doses to animals as parathion, and the class of cholinesterase inhibiting compounds manifests little tendency to chronic effect. Vigliani and Zurlo (1955) on the basis of cholinesterase reduction in workmen, consider 0.0007 mg./cu.m. a satisfactory threshold limit.

The most important effect of TEPP inhalation is the reduction of blood cholinesterase. The 0.05 mg./cu.m. threshold limit can be interpreted only by analogy with parathion. It appears low enough to prevent injury.

p-Tertiary butyl toluene. Hine et al. (1954) in repeated animal inhalations, found narcosis, respiratory tract irritation, liver and kidney changes, blood cell changes like those from benzene, and degenerations in spinal cord and brain. Rats are killed in one hour by about 900 ppm, and slight evidence of effect was found in animals repeatedly inhaling 25 ppm. Humans detect 5 ppm by odor, 80 ppm was unpleasantly irritating and some giddiness was noted at 160 ppm.

The most important effect of p-tertiary butyl toluene inhalation appears to be chronic toxicity, combining effect on blood cells, central nervous system, liver and kidney. The 10 ppm threshold limit can be interpreted from results of repeated animal inhalations and human sensory data. It appears to be low enough to prevent significant toxic effect.

1,1,2,2-Tetrachloroethane. Fairhall (1949, p. 440) concludes this is the most toxic chlorinated hydrocarbon, nine times as toxic as carbon tetrachloride. It is a narcotic and produces liver damage, polyneuritis and white blood cell changes. Elkins (1950, p. 139) refers to an unpublished report of illness from a concentration below 10 ppm. Smyth (1937-55) found rats survive four hours at 500 ppm but are killed by 1000 ppm.

The most important effect of 1,1,2,2-tetrachloroethane inhalation is chronic poisoning centering in the liver. The 5 ppm threshold limit can be interpreted from industrial experience. It is uncertain whether it is low enough to prevent some degree of injury.

Tetrahydrofuran. Lehman and Flury (1943, p. 269) report it a narcotic, irritating mucous membrane and injuring the kidneys. In animals 3400 ppm for eight hours daily for 20 days caused some mucous membrane irritation and light narcosis, with albuminuria, lung and kidney injury and lung irritation in one animal each. ACGIH (1955b) quotes John A. Zapp, Jr. to the effect that repeated inhalation of 200 ppm, then 400 ppm, slightly affected the pulse pressure of dogs, but resulted in no histopathology. Hoffmann and Oettell (1954) in rabbits and cats found some narcosis and mucosal irritation after six hours

inhalation of 3400 ppm. There was no liver or kidney injury from inhalation of even 60,000 ppm.

The most important effect of tetrahydrofuran inhalation is narcosis, with less important injury to liver and kidneys. The 200 ppm tentative threshold limit can be interpreted from results of repeated animal inhalation studies. It appears low enough to prevent injury.

Tetranitromethane. Sievers, Rushing, Gay and Monaco (1947) found in cats irritation of eyes, upper respiratory tract, lung edema, methemoglobinuria and injury to liver and kidney. Concentrations of 3.3 to 25.2 ppm were severely injurious, while 0.1 to 0.4 ppm caused only mild irritation in two exposures. Horn (1954) found in repeated inhalation of 6.35 ppm that some rats died from pneumonia. The effect on dogs was transient anorexia.

The most important effect of tetranitromethane inhalation is poisoning, accompanied by irritation. The 1 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent injury, but not to prevent all irritation.

Tetryl. Bergman (1952) summarizes ten years' experience in an ordnance plant with more than 1000 exposed workers and concentrations kept below 1.5 mg./cu.m. by constant diligence. Skin sensitization was frequent but no systemic poisoning was found. Hardy and Maloof (1950) report two fatal and eight non-fatal cases with liver injury the most prominent effect. There was upper respiratory tract irritation, very frequent skin sensitization and a few asthmatic sensitizations. Concentrations were as high as 17.7 mg./cu.m.

The most important effect of tetryl inhalation is chronic poisoning like that of trinitrotoluene, but the most frequent effect is sensitization. The 1.5 mg./cu.m. threshold limit can be interpreted from experience with exposed workmen. It is low enough to prevent injury but not sensitizations.

Thallium. Fairhall (1949) reviewing the literature, concludes that thallium has a chronic toxicity greater than that of lead. Useful quantitative data on inhalation appear to be lacking.

The most important effect of thallium inhalation is chronic poisoning. The 0.15 mg./cu.m. tentative threshold limit appears to be based on a quantitative analogy with lead. Its propriety cannot be judged.

Thiram. Meagre data have been found on this substance. Smyth (1937-55) found the rat oral LD_{50} to be 1.30 gm./kg. Rats survived four hours inhalation of a dense dust cloud, unmeasured but estimated to be at least 500 mg./cu.m., with no effect but some brief retardation of growth.

The most important effect of inhalation of thiram appears to be an ill-defined chronic toxicity. The 5 mg./cu.m. tentative threshold limit cannot be interpreted from published data found by the writer, but it appears to be reasonable.

Titanium dioxide. Fairhall (1949, p. 180) concludes titanium dioxide is chemically inert and is not toxic. Lenzi (1936) found a pneumoconiosis in guinea pig lungs after prolonged inhalation of high concentrations.

Inhaled titanium dioxide acts as an inert dust, with only a slight tendency to produce pneumoconiosis. The 15 mg./cu.m. threshold limit is an arbitrary figure uniformly applied to inert nuisance dusts. It appears low enough to prevent injury.

Toluene. Von Oettingen, Neal and Donahue (1942) in repeated exposure of animals, found no blood cell changes or other toxic effects at 800 ppm. Humans inhaling 200 ppm for an eight-hour period found the earliest signs of impaired coordination and lengthened reaction time, while the effects were more prominent and more prompt at 600 to 800 ppm. Fairhall (1949, p. 447) concludes it is a narcotic with irritating properties, but manifests no chronic effects. Elkins (1950, p. 108) cites Greenburg's examination of over 100 workers in atmospheres of 100 to 1100 ppm without marked symptoms. Smyth (1937-55) found rats survive four hours at 4000 ppm, but die from 16,000 ppm. Commercial toluene may contain significant amounts of benzene and may be more toxic than these data indicate.

The most important effect of toluene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalation, studies of human narcosis and examination of exposed workmen. It appears to be low enough to prevent all effects except the earliest signs of narcosis.

O-Toluidine. Henderson and Haggard (1943, p. 228) quote slight symptoms after several hours at 6 to 23 ppm, and for aniline they give 7 to 53 ppm. Fairhall (1949, p. 450) find symptoms are like those from aniline. Smyth (1937-55) found rats not killed by eight hours inhalation of substantially saturated vapors.

The most important effect of o-toluidine inhalation is poisoning like that from aniline. The 5 ppm threshold limit can be interpreted by analogy with aniline. It appears low enough to prevent injury.

Trichloroethylene. Morse and Goldberg (1943) found headache, nausea and dizziness common in industrial exposures well under the 200 ppm figure proposed by USPHS (1943) on the basis of European opinion. Adams, Spencer, Rowe, McCollister and Irish (1951) found no adverse effects from repeated inhalation by monkeys at 400 ppm, in rats and rabbits at 200 ppm, or in guinea pigs at 100 ppm. Single massive exposures kill by anesthesia. Even 3000 ppm inhaled repeatedly causes no more than very minor organic changes in the liver. They found almost identical effects from single and repeated inhalation of a single concentration, and concluded the effects are chiefly those of narcosis. Voluntary habituation has been found in industry.

The most important effect of trichloroethylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of repeated animal inhalations and observations in industry. It is not low enough to prevent significant narcosis.

Trichloronaphthalene. After extensive inhalation studies with rats, Drinker (1939) concluded injury is exclusively in the liver and that the permissible limit for repeated inhalation is 5 mg./cu.m. The solid material or its oil solution penetrates the skin and repeated contact leads to chloracne.

The most important effect of trichloronaphthalene inhalation is chronic poisoning centering in the liver. The 5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Trifluoromonobromomethane. ACGIH (1955b) quotes Chemical Corps Medical Laboratories Research Report No. 180, 1953. This shows that the material is narcotic at high concentrations, but that 23,000 ppm for 18 weeks had no toxic or lung injuring effects on dogs or rats.

The most important effects of trifluoromonobromomethane inhalation are narcosis and respiratory tract irritation. The 1000 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Trinitrotoluene. Von Oettingen et al (1944) in experiments on dogs found only tracheal irritation and some effects on red blood cells from daily intratracheal insufflation of 50 mg./kg. This daily dosage by mouth did not kill in three months but resulted in definite central nervous system, liver and blood cell effects. The material penetrates the skin. Eddy (1944) reports fatal aplastic anemia in three humans at concentrations of 1 to 3.5 mg./cu.m.

The most important effect of trinitrotoluene inhalation is chronic poisoning marked by central nervous system, liver and red blood cell changes. The 1.5 mg./cu.m. threshold limit can be interpreted from a report of industrial fatality. It is probably not low enough to prevent all injuries.

Turpentine. Smyth and Smyth (1928) found that repeated inhalation of 750 ppm did not injure animals. Nelson, Ege, Ross, Woodman and Silverman (1943) with unacclimated subjects found nose and throat irritation at 75 ppm, while 175 ppm was judged intolerable. Fairhall (1949, p. 464) notes irritation, narcosis, and kidney injury.

The most important effect of turpentine inhalation is narcosis, but irritation of the respiratory tract is more frequently encountered. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations and human sensory data. It is low enough to prevent injury.

Uranium (soluble). Uranium is a radiological hazard through emission of alpha particles, and a toxicological hazard primarily through injury to kidney tubules. Hodge, Stokinger and Neuman (1949) show that on inhalation, the toxicological hazard is much greater than the radiological hazard. In repeated animal experiments, they find that 0.5 mg./cu.m. allows a reasonable margin of safety over the level producing kidney injury.

The most important effect of inhalation of soluble uranium dusts is chronic poisoning, centering in the kidney. The 0.05

mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Uranium (insoluble). Hodge, Stokinger, Neuman, Bale and Brandt (1949) show that on inhalation, the toxicological action of uranium on the kidney is much more a hazard than the alpha radiation hazard of the uranium stored in the bones. In repeated animal experiments, they find 0.20 to 0.25 mg./cu.m. allows reasonable margin of safety over the level producing kidney injury.

The most important effect of inhalation of insoluble uranium dusts is chronic poisoning centering in the kidney. The 0.25 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Vanadium. Roshchin (1952) exposed rats to vanadium pentoxide fume of 0.3 to 0.5 mg./cu.m. for two hours every other day for three months, and to dust at 1 to 3 mg./cu.m. for one hour daily for four months. They lost some weight and had bloody nasal secretion. Some evidence of pulmonary edema was seen from the fume. Acutely 8 mg./cu.m. of the dust was injurious in one hour and 70 to 80 mg./cu.m. was lethal. The author suggests a threshold limit of 0.1 mg./cu.m. for fume and 0.5 mg./cu.m. for dust.

The most important effect of inhalation of vanadium dusts is bronchial and lung injury. The threshold limits of 0.5 mg./cu.m. for dust and 0.1 mg./cu.m. for fume can be interpreted from the results of repeated animal inhalations. They appear low enough to prevent injury.

Vinyl chloride. Patty, Yant and Waite (1930) found no serious disturbance in guinea pigs inhaling 5000 ppm for several hours. Higher concentrations produced only narcosis. In humans, 50,000 ppm is noticed as a slight odor and nose irritation, and dizziness is evident.

The most important effect of vinyl chloride inhalation is narcosis. The 500 ppm threshold limit can be interpreted from the results of single animal inhalations and human response. It appears low enough to prevent significant narcosis.

Warfarin. Saunders, Heisey, Goldstone and Bay (1955) found injection of 0.5 mg./kg. for five days killed most of a small group of rats, while a single injection of 100 mg./kg. caused only 30% mortality. One human fatality arose from the consumption of a total of 750 mg. over a 15-day period.

The most important effect of warfarin inhalation is chronic poisoning centering in the blood coagulation mechanism. The 0.5 mg./cu.m. tentative threshold limit cannot be interpreted in quantitative terms. It involves a maximum daily absorption of 5 mg., about one-tenth the amount which was fatal to one man.

Xylene. Nelson, Ege, Ross, Woodman and Silverman (1943) found 200 ppm definitely irritating to eye, nose and throat. Fairhall (1949, p. 468) concludes the effects are like those of toluene, narcosis without damage to red blood cells.

The most important effect of xylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from human sensory data. It is irritating to eye, nose and throat. There are no data to show whether or not significant narcosis occurs at this concentration, but since narcotic activity is greater than that of toluene it is to be anticipated.

Zinc oxide fume. Drinker, Thomson and Finn (1927b) reported that experimental fume fever from zinc oxide in man results from excessive inhalation, but does not occur below 15 mg./cu.m. In industry it was found that 14 mg./cu.m. caused no reaction after eight hours, and in the laboratory 45 mg./cu.m. was without effect in 20 minutes. This condition is a transient fever with chills, muscular pains, nausea and vomitting. An immunity is apparently built up.

The most important effect of zinc oxide fume inhalation is transient metal fume fever. The 15 mg./cu.m. threshold limit can be interpreted from the results of extensive human experiment and experience in industry. It is low enough to prevent injury.

Zirconium. ACGIH (1955b) quotes unpublished data from the University of Rochester A.E.C. Project. Four species of animals inhaled the following, all expressed in terms of contained zirconium: Zirconium oxide, 1.5 micron dust, 75 mg./cu.m. for 30 days, 11 mg./cu.m. for 60 days, 3.5 mg./cu.m. for one year; zirconium tetrachloride, 0.6 micron dust, 6 mg./cu.m. for 60 days, 3.5 mg./cu.m. for a year. Only the higher concentration of tetrachloride had any effect, presumably due to liberated hydrochloric acid.

The effect of zirconium dust inhalation is that of an inert nuisance dust when insoluble, and possibly bronchial and lung irritation when soluble. The 5 mg./cu.m. tentative threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Bibliography

ACGIH (1947): 1947 M.A.C. values. *Ind. Hyg. Newsletter*, 7:15-16, August.

ACGIH (1948): Threshold limit values adopted at April, 1948 meeting (privately circulated).

ACGIH (1949): Threshold limit values adopted at April 1949 meeting (privately circulated).

ACGIH (1950): Threshold limit values. *Arch. Ind. Hyg. & Occup. Med.*, 2:98-100.

ACGIH (1951): Threshold limit values for 1951. *Arch. Ind. Hyg. & Occup. Med.*, 4:398-400.

ACGIH (1952): Threshold limit values for 1952. *Arch. Ind. Hyg. & Occup. Med.*, 6:178-180.

ACGIH (1953): Threshold limit values for 1953. *Arch. Ind. Hyg. & Occup. Med.*, 8:296-298.

ACGIH (1953b): Privately circulated document giving support for many earlier adopted threshold limits, and those proposed in 1953.

ACGIH (1954): Threshold limit values for 1954. *Arch. Ind. Hyg. & Occup. Med.*, 9:530-534.

ACGIH (1954b): Privately circulated document giving support for threshold limits newly proposed in 1954.

ACGIH (1955): Threshold limit values for 1955. *AMA Arch. Ind. Health*, 11:521-524.

ACGIH (1955b): Privately circulated document giving support for threshold limits newly proposed in 1955.

ACGIH (1956): Proposed threshold limit values for 1956, presented April 23, 1956 to the Philadelphia, annual meeting. Privately circulated in advance of publication.

ADAMS, E. M.; SPENCER, H. C., and IRISH, D. D. (1940): The acute vapor toxicity of allyl chloride. *J. Ind. Hyg. & Tox.*, 22:79-86.

ADAMS, E. M.; SPENCER, H. C.; ROWE, V. K., and IRISH, D. D. (1950): Vapor toxicity of 1,1,1-trichloroethane determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 1:225-236.

ADAMS, E. M.; SPENCER, H. C.; ROWE, V. K.; MCCOLLISTER, D. D. and IRISH, D. D. (1951): Vapor toxicity of trichloroethylene determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 4:469-481.

ADAMS, E. M.; SPENCER, H. C.; ROWE, V. K.; MCCOLLISTER, D. D., and IRISH, D. D. (1952): Vapor toxicity of carbon tetrachloride determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 6:50-66.

AERO MEDICAL ASSOCIATION (1953), Committee on Aviation Toxicology: *Aviation Toxicology*, Blakiston, New York.

AHLMARK, A. (1948): Poisoning by methyl mercury compounds. *Brit. J. Ind. Med.*, 5:117-119.

ALVAREZ, W. C., and HYMAN, S. (1953): Absence of toxic manifestations in workers exposed to chlordane. *Arch. Ind. Hyg. & Occup. Med.*, 8:480-483.

AMA (1951) Council on Pharmacy and Chemistry: Toxic effects of technical benzene hexachloride and its principal isomers. *J.A.M.A.*, 147:571-574.

AMDUR, M. O.; SILVERMAN, L., and DRINKER, P. (1952): Inhalation of sulfuric acid mist by human subjects. *Arch. Ind. Hyg. & Occup. Med.*, 6:305-313.

ASHE, W. F.; LARGENT, E. J.; DUTRA, F. R.; HUBBARD, D. M., and BLACKSTONE, M. (1953): Behavior of mercury in the animal organism following inhalation. *Arch. Ind. Hyg. & Occup. Med.*, 7:19-43.

Baltimore City Health Department (1943), Division of Industrial Hygiene: *Baltimore Health News*, 20: September, 1943.

BARNES, J. M. (1953): Toxic hazards of certain pesticides to man. World Health Organization, Geneva. Monograph 16.

BARTHELEMY, H. L. (1939): Ten years experience with industrial hygiene in connection with the manufacture of viscose rayon. *J. Ind. Hyg. & Tox.*, 21:141-151.

BERGMAN, B. B. (1952): Tetryl toxicity. *Arch. Ind. Hyg. & Occup. Med.*, 5:10-20.

BLOOMFIELD, J. J., and BLUM, W. (1928): Health hazards in chromium plating. U.S. Public Health Service, *Pub. Health Rpts.*, 43:2330-2351.

BRADLEY, W. R., and FREDRICK, W. G. (1941): The toxicity of antimony: Animal studies. *Ind. Med.*, 10, Ind. Hyg. Sec., 2:15-22.

BRANDT, A. D. (1947): Industrial Health Engineering. Wiley, New York.

BRIEGER, H., and HODES, W. A. (1951): Toxic effects of exposure to vapors of aliphatic amines. *Arch. Ind. Hyg. & Occup. Med.*, 3:287-291.

BRIEGER, H.; SEMISCH, C. W.; STASNEY, J., and PIATNEK, D. A. (1954): Industrial antimony poisoning. *Ind. Med.*, 23:521-523.

BROWN, H. V., and BUSH, A. F. (1950): Parathion inhibition of cholinesterase. *Arch. Ind. Hyg. & Occup. Med.*, 1:633-636.

BUCHAN, R. F. (1947): Industrial selenosis. *Occup. Med.*, 3:439-436.

CAMERON, G. R.; THOMAS, J. C.; ASHMORE, S. A.; WARREN, E. H.; BUCHAN, J. L., and MCKENNY-HUGHES, A. W. (1937): The toxicity of certain chlorine derivatives of benzene, with special reference to o-dichlorobenzene. *J. Path. Bact.*, 44:281-296.

CARPENTER, C. P. (1937). The chronic toxicity of tetrachloroethylene. *J. Ind. Hyg. & Tox.*, 19:323-336.

CARPENTER, C. P.; SHAFFER, C. B.; WEIL, C. S., and SMYTH, H. F., JR. (1944): Studies on the inhalation of 1:3-Butadiene. *J. Ind. Hyg. & Tox.*, 26:69-78.

CARPENTER, C. P.; SMYTH, H. F., JR., and SHAFFER, C. B. (1948): The acute toxicity of ethylene imine to small animals. *J. Ind. Hyg. & Tox.*, 30:2-6.

CARPENTER, C. P.; WEIL, C. S.; POZZANI, U. C., and SMYTH, H. F., JR. (1950): Comparative acute and subacute toxicities of allethrin and pyrethrins. *Arch. Ind. Hyg. & Occup. Med.*, 2:420-432.

CARPENTER, C. P.; POZZANI, U. C., and WEIL, C. S. (1953): Toxicity and hazard of diisobutyl ketone vapors. *Arch. Ind. Hyg. & Occup. Med.*, 8:377-381.

Committee on Chemical Agents (1949). Report of the panel on Environmental Agents, Ninth Annual Congress on Industrial Health, Chicago, January 18-19, 1949. Published in *Arch. Ind. Hyg. & Occup. Med.*, 1:601-624, 1950.

COMSTOCK, C. C.; MACNAMEE, J. K.; OZBURN, E. E.; FOGLEMAN, R. W., and OBERST, F. W. (1952): Monochloromonobromomethane; inhalation toxicity, pathology and symptomatology in rats and mice. Chemical Corps Medical Laboratories, Research Report No. 113.

COMSTOCK, C. C., and OBERST, F. W. (1953): The median detectable concentration of diborane, pentaborane and decaborane by odor for man. Chemical Corps Medical Laboratories, Research Report No. 206.

COMSTOCK, C. C.; LAWSON, L. H.; GREENE, E. A., and OBERST, F. W. (1954): Inhalation toxicity of hydrazine vapor. *Arch. Ind. Hyg. & Occup. Med.*, 10:476-490.

COOK, W. A. (1945): Maximum allowable concentrations of industrial atmospheric contaminants. *Ind. Med.*, 14:936-946.

CULVER, D.; CAPLAN, P., and BATCHELOR, G. S. (1956): Studies of human exposure during aerosol application of malathion and chlorthion. *AMA Arch. Ind. Health*, 13:37-50.

DEICHMANN, W. B.; KITZMILLER, K. V., and WITHERUP, S. (1944): Phenol studies VII. Chronic phenol poisoning, with special reference to the effects upon experimental animals of the inhalation of phenol vapor. *Am. J. Clin. Path.*, 14:273-277.

DERNEHL, C. U.; NAU, C. A., and SWEETS, H. H. (1945): Animal studies on the toxicity of inhaled antimony trioxide, *J. Ind. Hyg. & Tox.*, 27:256-262.

DERNEHL, C. U. (1951): Clinical experiences with exposures to ethylene amines. *Ind. Med. & Surg.*, 20:541-546.

DIEKE, S. H., and RICHTER, C. P. (1946): Comparative assays of rodenticides on wild Norway rats. U.S. Public Health Service, *Pub. Health Rpts.*, 61:672-679.

DIERKER, H., and BROWN, P. G. (1944): Study of a fatal case of ethylene chlorhydrin poisoning. *J. Ind. Hyg. & Tox.*, 26:277-279.

DONLEY, D. E. (1936): Toxic encephalopathy and volatile solvents in industry. *J. Ind. Hyg. & Tox.*, 18:571-577.

DRINKER, C. K. (1939): Further observations on the possible systemic toxicity of certain of the chlorinated hydrocarbons. *J. Ind. Hyg. & Tox.*, 21:155-159.

DRINKER, P.; THOMSON, R. M., and FINN, J. L. (1927): Metal fume fever. III. The effects of inhaling magnesium oxide fume. *J. Ind. Hyg.*, 9:187-192.

DRINKER, P.; THOMSON, R. M., and FINN, J. L. (1927b): Metal fume fever. IV. Threshold doses of zinc oxide, preventive measures and the chronic effects of repeated exposures. *J. Ind. Hyg.*, 9:331-345.

DRINKER, P.; YAGLOU, C. P., and WARREN, M. F. (1943): The threshold toxicity of gasoline vapor. *J. Ind. Hyg. & Tox.*, 25:225-232.

DUDLEY, H. C., and MILLER, J. W. (1941): Effects of subacute exposure to hydrogen selenide. *J. Ind. Hyg. & Tox.*, 23:470-477.

DUDLEY, H. C., and NEAL, P. A. (1942): Toxicology of acrylonitrile. I. A study of the acute toxicity. *J. Ind. Hyg. & Tox.*, 24:27-36.

DUDLEY, H. C.; SWEENEY, T. R., and MILLER, J. W. (1942). Toxicology of acrylonitrile. II. Studies of effects of daily inhalation. *J. Ind. Hyg. & Tox.*, 24:255-258.

EDDY, J. H., JR. (1944): Aplastic anemia following trinitrotoluene exposure. *J.A.M.A.*, 125:1169-1172.

ELKINS, H. B. (1950): The chemistry of industrial toxicology. Wiley, New York.

FAIRHALL, L. T., and MILLER, J. W. (1941): A study of the relative toxicity of the molecular components of lead arsenate. U. S. Public Health Service, *Pub. Health Rpts.*, 56:1610-1625.

FAIRHALL, L. T.; MILLER, J. W., and WEAVER, F. L. (1943): The effect of arsenates on the storage of lead. U.S. Public Health Service, *Pub. Health Rpts.*, 58:955-959.

FAIRHALL, L. T.; DUNN, R. C.; SHARPLESS, N. E., and PRITCHARD, E. A. (1945): The toxicity of molybdenum. U.S. Public Health Service, *Pub. Health Bull.* 293.

FAIRHALL, L. T. (1949): Industrial Toxicology. Williams & Wilkins, Baltimore.

FAIRLEY, A.; LINTON, E. C., and FORD-MOORE, A. H. (1934): The toxicity to animals of 1,4-dioxane. *J. Hyg.*, 34:486-501.

FEINER, B.; BURKE, W. J., and BALIFF, J. (1946): An industrial hygiene survey of an onion dehydrating plant. *J. Ind. Hyg. & Tox.*, 28:278-279.

FIELDNER, A. C.; KATZ, S. H., and KINNEY, S. P. (1921): Gas masks for gases met in fighting fires. U.S. Bur. Mines Tech. Paper 248.

FIELDNER, A. C.; SAYERS, R. R.; YANT, W. P.; KATZ, S. H.; SHOHAN, J. B., and LEITCH, R. D. (1931): Warning agents for fuel gases. U.S. Bur. Mines, Monograph #4.

FINE, E. A., and WILLS, J. H. (1950): Pharmacologic studies on furfuryl alcohol. *Arch. Ind. Hyg. & Occup. Med.*, 1:625-632.

FITZHUGH, O. G., and NELSON, A. A. (1947): Chronic oral toxicity of alpha-naphthyl-thiourea. *Proc. Soc. Exper. Biol. Med.*, 64:305-310.

FLINN, R. H.; NEAL, P. A., and FULTON, W. B. (1941): Industrial manganese poisoning. *J. Ind. Hyg. & Tox.*, 23:374-387.

FLURY, F., and ZERNIK, F. (1931): Schädliche Gase und Dämpfe. J. Springer, Berlin.

GOLDBLATT, M. W. (1944): Toxic effects of ethylene chlorhydrin. II—Experimental. *Brit. J. Ind. Med.*, 1:213-223.

GOLDBLATT, M. W., and CHIESMAN, W. E. (1944): Toxic effects of ethylene chlorhydrin. I—Clinical. *Brit. J. Ind. Med.*, 1:207-213.

GRAY, E. LEB., MACNAMEE, J. K., and GOLDBERG, S. B. (1952): Toxicity of NO_2 vapors at very low levels. *Arch. Ind. Hyg. & Occup. Med.*, 6:20-21.

GREENBERG, L. A., and LESTER, D. (1950): The toxicity of sulfurpentafluoride. *Arch. Ind. Hyg. & Occup. Med.* 2:350-352.

GREENBERG, L.; MAYERS, M. R.; GOLDWATER, L. J.; BURKE, W. J., and MOSKOWITZ, S. (1938): Health hazards in the manufacture of fused collars. I—Exposure to ethylene glycol monomethyl ether. *J. Ind. Hyg. & Tox.*, 20:134-147.

HAAG, H. B.; FINNEGAN, J. K.; LARSON, P. S.; RIESE, W., and DREYFUSS, M. L. (1950): Comparative chronic toxicities for warm-blooded animals. of 2,2-bis-(p-chlorophenyl)-1,1,1-trichloroethane (DDT) and 2,2-bis-(p-methoxyphenyl)-1,1,1-trichloroethane (DMDT, methoxychlor). *Arch. Intern. Pharmacodynamie*, 83:491-504.

HAGGARD, H. W.; GREENBERG, L. A., and TURNER, J. M. (1944): The physiological principles governing the action of acetone, together with determination of toxicity. *J. Ind. Hyg. & Tox.*, 26:133-151.

HAGGARD, H. W.; MILLER, D. P., and GREENBERT, L. A. (1945): The amyl alcohols and their ketones: their metabolic fates and comparative toxicities. *J. Ind. Hyg. & Tox.*, 27:1-14.

HANZLIK, P. J. (1923): Toxicity and actions of the butyl amines. *J. Pharm. Exptl. Therap.*, 20:435-449.

HARDY, H. L., and MALOOF, C. C. (1950): Evidence of systemic effect of tetryl. *Arch. Ind. Hyg. & Occup. Med.*, 1:545-555.

HAYHURST, E. R. (1936): Poisoning by petroleum distillates. *Ind. Med.*, 5:53-63.

HEMEON, W. C. L. (1955). Plant and process ventilation. The Industrial Press, New York.

HENDERSON, Y.; HAGGARD, H. W.; TEAGUE, M. C.; PRINCE, A. L., and WUNDERLICH, R. M. (1921): Physiological effects of automobile exhaust gas and standards of ventilation for brief exposure. *J. Ind. Hyg.*, 3:79-137.

HENDERSON, Y., and HAGGARD, H. W. (1943): Noxious Gases, 2nd ed. Reinhold, New York.

HEPPEL, L. A.; NEAL, P. A.; PERRIN, T. L.; ORR, M. L., and PORTERFIELD, V. T. (1944): Toxicology of dichloromethane. *J. Ind. Hyg. & Tox.*, 26:8-21.

HEPPEL, L. A.; NEAL, P. A.; HIGHMAN, B., and PORTERFIELD, V. T. (1946): Toxicology of 1,2-dichloropropane. *J. Ind. Hyg. & Tox.*, 28:1-8.

HINE, C. H., *et al.* (1954): Toxicological studies on p-tertiary butyl toluene. *Arch. Ind. Hyg. & Occup. Med.*, 9:227-244.

HODGE, H. C.; STOKINGER, H. E., and NEUMAN, W. F. (1949): Suggested maximum concentration of soluble uranium compounds in air. AECD 2785, n.d., decl. Dec. 1949.

HODGE, H. C.; STOKINGER, H. E.; NEUMAN, W. F.; BALE, W. F., and BRANDT, A. E. (1949): Suggested maximum allowable concentration of insoluble uranium compounds in air. AECD 2784, n.d., decl. 1949.

HODGE, H. C.; MAYNARD, E. A., and BLANCHET, H. J., JR. (1952): Chronic oral toxicity tests of Methoxychlor in rats and dogs. *J. Pharm. Exptl. Therap.*, 104:60-66.

HODGE, H. C.; MAYNARD, E. A.; DOWNS, W.; BLANCHET, H. J., JR., and JONES, C. K. (1952): Acute and Short-term oral toxicity tests of ferric dimethyldithiocarbamate (Ferbam) and zinc dimethyldithiocarbamate (Ziram). *J. Amer. Pharm. Assn., Sci. ed*, 41:662-665.

HODGE, H. C.; MAYNARD, E. A.; HURWITZ, L., DISTEFANO, V.; DOWNS, W. L.; JONES, C. K., and BLANCHET, H. J., JR. (1954): Studies of the toxicity and of the enzyme kinetics of ethyl p-nitrophenyl thionobenzene phosphonate. *J. Pharm. Exptl. Therap.*, 112:29-39.

HOFMANN, and OETTEL, H. (1954): Toxicity of tetrahydrofuran. *Naunyn-Schmiedebergs Arch. Exptl. Pathol. Pharmakol;* 222:233-235. (From *Chem. Abstracts*, 48:7788, 1954).

HOLLINGSWORTH, R. L.; ROWE, V. K.; OYEN, F.; MCCOLLISTER, D. D., and SPENCER, H. C. (1956): Toxicity of ethylene oxide determined on experimental animals. *Arch. Ind. Health*, 13:217-227.

HORN, H. J. (1954): Inhalation toxicology of tetranitromethane. *Arch. Ind. Hyg. & Occup. Med.*, 10:213-222.

HORN, H. J., and WEIR, R. J. (1955): Inhalation toxicology of chlorine trifluoride. *Arch. Ind. Health*, 12:515-521.

HORN, H. J., and WEIR, R. J. (1956): Inhalation toxicology of chlorine trifluoride. *Arch. Ind. Health*, 13:340-345.

HUEPER, W. C. (1950): Carcinogens and carcinogenesis. *Am. J. Med.*, 8:355-371.

I.L.O. (1930): Occupation and health, 2nd vol. International Labour Office, Geneva.

INGLE, L. (1953): The toxicity of chlordane vapors. *Science*, 118:213-214.

INGRAM, F. R. (1951): Methyl bromide fumigation and control in the date-packing industry. *Arch. Ind. Hyg. Occup. Med.*, 4:193-198.

IRISH, D. D.; ADAMS, E. M.; SPENCER, H. C., and ROWE, V. K. (1940): The response attending exposure of laboratory animals to vapors of methyl bromide. *J. Ind. Hyg. & Tox.*, 22:218-230.

JACOBSON, K. H.; HACKLEY, E. B., and FEINSILVER, L. (1956): The toxicity of inhaled ethylene oxide and propylene oxide vapors. *Arch. Ind. Health*, 13:237-244.

JOHNSON, G. A.; FLETCHER, J. H.; NOLAN, K. G., and CASSADAY, J. T. (1952): Decreased toxicity and cholinesterase inhibition in a new series of dithiophosphates. *J. Econ. Entomol.*, 45:279-283.

KEHOE, R. A.; MACHLE, W. F.; KITZMILLER, K., and LEBLANC, T. J. (1932). On the effects of prolonged exposure to sulfur dioxide. *J. Ind. Hyg.*, 14:159-173.

KEHOE, R. A.; DEICHMANN-GRUEBLER, W., and KITZMILLER, K. V. (1939): Toxic effects upon rabbits of pentachlorophenol and sodium pentachlorophenate. *J. Ind. Hyg. & Tox.*, 21:160-172.

KINCAID, J. F.; STRONG, J. S., and SUNDERMAN, F. W. (1953): Nickel poisoning. I—Experimental study of the effects of acute and subacute exposure to nickel carbonyl. *Arch. Ind. Hyg. & Occup. Med.*, 8:48-60.

KRACKOW, E. H. (1953): Toxicity and health hazards of boron hydrides. *Arch. Ind. Hyg. & Occup. Med*, 8:335-339.

LACKEY, R. W. (1949): Observations on the acute and chronic toxicity of toxaphene in the dog. *J. Ind. Hyg. & Tox.*, 31:117-120.

LARGENT, E. J. (1952): Rates of elimination of fluoride stored in the tissues of man. *Arch. Ind. Hyg. & Occup. Med.*, 6:37-42.

LEHMAN, A. J. (1949): Pharmacological considerations of insecticides. *Assoc. Food Drug Off. of U.S.,Q. Bull.* 13:65-70.

LEHMANN, K. B. (1886): Experimentelle Studien über den Einfluss technisch und hygienisch wichtiger Gase und Dämpfe auf den Qrganismus. *Arch. f. Hyg.*, 5:68.

LEHMANN, K. B., and FLURY, F. (1943): Toxicology and hygiene of industrial solvents. Translated by E. King and H. F. Smyth, Jr. Williams and Wilkins, Baltimore.

LENZI, L. (1936): Pneumoconiosis caused by titanium. *Rass. Med. Applicata Lavoro Ind.;* 7:301-318.

LESTER, D., and GREENBERG, L. A. (1950): The toxicity of sulfur hexafluoride. *Arch. Ind. Hyg. & Occup. Med.*, 2:348-349.

LUNDGREN, K-D., and SWENSSON, A. (1949): Occupational poisoning by alkyl mercury compounds. *J. Ind. Hyg. & Tox.*, 31:190-200.

MACHLE, W.; SCOTT, E. W., and TREON, J. (1939): The phyisological response to isopropyl ether and to a mixture of isopropyl ether and gasoline. *J. Ind. Hyg. & Tox.*, 21:72-96.

MACHLE, W., and EVANS, E. E. (1940): Exposure to fluorine in industry. *J. Ind. Hyg. & Tox.* 22:213-217.

MACHLE, W.; SCOTT, E. W., and TREON, J. (1940): The physiological response of animals to some simple mononitroparaffins and to certain derivatives of these compounds. *J. Ind. Hyg. & Tox.*, 22:315-332.

MACHLE, W.; KITZMILLER, K. V.; SCOTT, E. W., and TREON, J. F. (1942): The effect of inhalation of hydrogen chloride. *J. Ind. Hyg. & Tox.*, 24:222-225.

MACHLE, W.; SCOTT, E. W.; TREON, J. F.; HEYROTH, F. F., and KITZMILLER, K. V. (1945): The physiological response of animals to certain chlorinated mononitroparaffins. *J. Ind. Hyg. & Tox.*,, 27:95-102.

MCCLOSKY, W. T.; SMITH, M. I., and LILLIE, R. D. (1945): Studies on the pharmacologic action and the pathology of alphanaphthylthiourea (ANTU) U.S. Public Health Service, *Pub. Health Rpts.*, 60:1101-1113.

MCCORD, C. P. (1932): The toxicity of allyl alcohol. *J.A.M.A.*, 98:2269-2270.

MCGEE, L. C. (1955): Chlorinated insecticides; toxicity for man. *Ind. Med. and Surg.*, 24:101-109.

MCLAUGHLIN, R. S. (1946): Chemical burns of the human cornea. *Am. J. Ophthal.*, 29:1355-1362.

MCNALLY, W. D. (1937): Toxicology. Industrial Medicine Pub. Co. Chicago, Illinois.

MORSE, K. M., and GOLDBERG, L. (1943): Chlorinated solvent exposures. *Ind. Med.*, 12:706-713.

NAU, C. A. (1948): The accidental generation of arsine gas in an industry. *Southern Med. J.*, 41:341-344.

NEAL, P. A.; FLINN, R. H.; EDWARDS, T. I.; REINHART, W. H.; HOUGH, J. W.; DALLAVALLE, J. M.; GOLDMAN, F. H.; ARMSTRONG, D. W.; GRAY, A. S.; COLEMAN, A. L., and POSTMAN, B. F. (1941): Mercurialism and its control in the felt hat industry. U.S. Public Health Service, *Pub. Health Bull.* 263.

NELSON, K. W.; EGE, J. F., JR.; ROSS, M.; WOODMAN, L. E., and SILVERMAN, L. (1943). Sensory response to certain industrial solvent vapors. *J. Ind. Hyg. Tox.*, 25:282-285.

NUCKOLLS, A. H. (1933): The comparative life, fire and explosion hazards of common refrigerants. Underwriters' Lab. Report, Miscellaneous Hazards 2375.

OBERST, F. W.; COMSTOCK, C. C., and MCGRATH, F. P., (1951): Comparative toxicity of halogenated hydrocarbons proposed for fire extinguishants. *Federation Proc.*, 10:328.

OBERST, F. W.; COMSTOCK, C. C., and HACKLEY, E. B. (1954): Inhalation toxicity of 90% hydrogen peroxide vapor. *Arch. Ind. Hyg. & Occup. Med.*, 10:319-327.

OBERST, F. W.; HACKLEY, E. B., and COMSTOCK, C. C. (1956): Chronic toxicity of aniline vapor (5 ppm) by inhalation. *Arch. Ind. Health*, 13: 379-384.

OGLESBY, F. L. (1956): Personal communication to H. F. Smyth Jr., May 18.

PARSONS,, C. E., and PARSONS, M. E. M. (1938): Toxic encephalopathy and granulopenic anemia due to volatile solvents in industry. *J. Ind. Hyg. & Tox.*, 20:124-133.

PATTY, F. A., and YANT, W. P. (1929): Odor intensity and symptoms produced by commercial propane, butane, pentane, hexane and heptane vapor. U.S. Bur. Mines, Rpts. Investigations 2979.

PATTY, F. A.; YANT, W. P., and WAITE, C. P. (1930): Acute response of guinea pigs to vapors of some new commercial organic compounds. V. Vinyl chloride. U.S. Public Health Service, *Pub. Health Rpts.*, 45:1963-1971.

PATTY, F. A.; SCHRENK, H. H., and YANT, W. P. (1935): Acute response of guinea pigs to vapors of some new

commercial organic compounds. VIII—Butanone. U.S. Public Health Service, *Pub. Health Rpts.*, 50:1217-1228.

PATTY, F. A.; YANT, W. P., and SCHRENK, H. H. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. XI—Secondary amyl acetate. U.S. Public Health Service, *Pub. Health Rpts.*, 51:811-819.

PATTY, F. A. (1948-9): Industrial hygiene and toxicology, 2 vols. Interscience, New York.

POLLOCK, L. J., FINKELMAN, I., and ARIEFF, A. J. (1943): Toxicity of pyridine in man. *Arch. Internal Med.*, 71:95-106.

POZZANI, U. C.; WEIL, C. S., and CARPENTER, C. P. (1949), Subacute vapor toxicity and range-finding data for ethyl acrylate. *J. Ind. Hyg. & Tox.*, 31:311-316.

POZZANI, U. C., and CARPENTER, C. P. (1951): Response of rodents to repeated inhalation of vapors of tetraethyl orthosilicate. *Arch. Ind. Hyg. & Occup. Med.*, 4:465-468.

POZZANI, U. C., and CARPENTER, C. P. (1954): Response of rats to repeated inhalation of ethylene diamine vapors. *Arch. Ind. Hyg. & Occup. Med.*, 9:223-226.

PRINCI, F., and SPURBECK, G. H. (1951): A study of workers exposed to the insecticides chlordan, aldrin, dieldrin. *Arch. Ind. Hyg. & Occup. Med.*, 3:64-72.

PRODAN, L. (1932): Cadmium poisoning. *J. Ind. Hyg.*, 14:174-196.

RILEY, E. C., and GOLDMAN, F. H. (1937): Control of chromic acid mists from plating tanks. U.S. Public Health Service. *Pub. Health Rpts.*, 52:172-174.

ROHOLM, K. (1937): Fluorine intoxication. Lewis & Co., London.

ROSHCHIN, I. V. (1952): Hygienic characteristics of industrial vanadium aerosol. *Gig. i. Sanit.*, 11:49-53.

ROWE, V. K.; SPENCER, H. C., and BASS, S. L. (1948): Toxicological studies on certain commercial silicones and hydrolyzable silane intermediates. *J. Ind. Hyg. & Tox.*, 30:332-352.

ROWE, V. K.; SPENCER, H. C.; MCCOLLISTER, D. D.; HOLLINGSWORTH, R. L., and ADAMS, E. M. (1952): Toxicity of ethylene dibromide determined on experimental animals. *Arch. Ind. Hyg. & Occup. Med.*, 6:158-173.

ROWE, V. K.; MCCOLLISTER, D. D.; SPENCER, H. C.; ADAMS, E. D., and IRISH, D. D. (1952): Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. *Arch. Ind. Hyg. & Occup. Med.*, 5:566-579.

ROWE, V. K., and HYMAS, T. A. (1954): Summary of toxicological information on 2,4-D and 2,4,5-T type herbicides and an evaluation of the hazards to livestock associated with their use. *Am. J. Vetern. Res.*, 15:622-629.

ROWE, V. K.; HOLLINGSWORTH, R. L.; OYEN, F.; MCCOLLISTER, D. D., and SPENCER, H. C. (1956): Toxicity of propylene oxide determined on experimental animals. *Arch. Ind. Health*, 13:228-236.

ROZENDAAL, H. M. (1951): Clinical observations on the toxicology of boron hydrides. *Arch. Ind. Hyg. & Occup. Med.*, 4:257-260.

RUSSELL, A. E.; JONES, R. R.; BLOOMFIELD, J. J.; BRITTEN, R. H., and THOMPSON, L. R. (1933): Lead poisoning in a storage-battery plant. U. S. Public Health Service, *Pub. Health Bull. 205.*

SAUNDERS, J. P.; HEISEY, S. R.; GOLDSTONE, A. D., and BAY, E. C. (1955): Comparative toxicities of Warfarin and some 2-acyl-1,3-indandiones in rats. *Agr. Food Chem.*, 3:762-765.

SAYERS, R. R.; FIELDNER, A. C.; YANT, W. P., and THOMAS, B. G. H. (1927): Experimental studies on the effect of ethyl gasoline and its combustion products. U.S. Bur. Mines, Monograph No. 2.

SAYERS, R. R.; YANT, W. P.; LEVY, E., and FULTON, W. B. (1929): Effects of repeated daily exposure of several hours to small amounts of automobile exhaust gas. U.S. Public Health Service, *Pub. Health Bull. 186.*

SAYERS, R. R.; YANT, W. P.; THOMAS, B. G. H., and BERGER, L. B. (1929): Physiological response attending exposure to vapors of methyl bromide, methyl chloride, ethyl bromide and ethyl chloride. U.S. Public Health Service, *Pub. Health Bull. 185.*

SAYERS, R. R.; FIELDNER, A. C.; YANT, W. P.; LEITCH, R. D., and PEARCE, S. J. (1930): Use of ethyl mercaptan to detect leaks in natural-gas distribution systems. U.S. Bur. Mines Rpts., Investigations 3007.

SAYERS, R. R.; YANT, W. P.; CHORNYAK, J., and SHOAF, H. W. (1930): Toxicity of dichlorodifluoromethane, a new refrigerant. U.S. Bur. Mines Rpts., Investigations 3013.

SAYERS, R. R.; SCHRENK, H. H., and PATTY, F. A. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. XII—Normal butyl acetate. U.S. Public Health Service, *Pub. Health Rpts.*, 51:1229-1236.

SAYERS, R. R.; YANT, W. P.; SCHRENK, H. H.; CHORNYAK, J.; PEARCE, S. J.; PATTY, F. A., and LINN, J. G. (1942): Methanol poisoning. I—Exposure of dogs to 450-500 ppm methanol vapor in air. U.S. Bur. Mines Rpts., Investigations 3617.

SCHRENK, H. H.; PATTY, F. A., and YANT, W. P. (1933): Acute response of guinea pigs to vapors of some new commercial organic compounds. VII—Dichloroethyl ether. U.S. Public Health Service, *Pub. Health Rpts.*, 48:1389-1398.

SCHRENK, H. H.; YANT, W. P.; CHORNYAK, J., and PATTY, F. A. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. XIII—Methyl formate. U.S. Public Health Service, *Pub. Health Rpts.*, 51:1329-1337.

SCHRENK, H. H.; YANT, W. P., and PATTY, F. A. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. X—Hexanone (methyl butyl ketone). U.S. Public Health Service, *Pub. Health Rpts.*, 51:624-631.

SEXTON, R. J., and HENSON, E. V. (1950): Experimental ethylene oxide human skin injuries. *Arch. Ind. Hyg. & Occup. Med.*, 2:549-564.

SIEVERS, R. F., EDWARDS, T. I.; MURRAY, A. L., and SCHRENK, H. H. (1942): Effects of exposure to known concentrations of CO. *J.A.M.A.*, 118:585-588.

SIEVERS, R. F.; RUSHING, E.; GAY, H., and MONACO, A. R. (1947): Toxic effects of tetranitromethane, a contaminant in crude TNT. U.S. Public Health Service, *Pub. Health Rpts.*, 62:1048-1061.

SILVER, S. D., and MCGRATH, F. P. (1948): A comparison of acute toxicities of ethylene imine and ammonia to mice. *J. Ind. Hyg. & Tox.*, 30:7-9.

SILVERMAN, L., SCHULTE, H. F.; and FIRST, M. W. (1946): Further studies on sensory response to certain industrial solvent vapors. *J. Ind. Hyg. & Tox.*, 28:262-266.

SILVERMAN, L.; WHITTENBERGER, J. L., and MULLER, J. (1949): Physiological response of man to ammonia in low concentrations. *J. Ind. Hyg. & Tox.*, 31:74-78.

SJOBERG, S-G. (1951): Health hazards in the production and handling of vanadium pentoxide. *Arch. Ind. Hyg. & Occup. Med.*, 3:631-646.

SKINNER, J. B. (1947): Toxicity of 2-Nitropropane. *Ind. Med.*, 16:441-443.

SKLYANSKAYA, R. M., and RAPPAPORT, J. L. (1935): Chronic chlorine poisoning of rabbits with small doses of chlorine, and the development of the fetus in chlorine-poisoned rabbits. *Arch. exptl. Path. Pharmakol.*, 177:276-287.

SMITH, W. W., and VON OETTINGEN, W. F. (1947): The acute and chronic toxicity of methyl chloride. *J. Ind. Hyg. & Tox.*, 29:47-52.

SMYTH, H. F., and SMYTH, H. F., JR. (1928): Inhalation experiments with certain lacquer solvents. *J. Ind. Hyg.*, 10:261-271.

SMYTH, H. F., JR. (1937-55): Unpublished work by Chemical Hygiene Fellowship, Mellon Institute, Pittsburgh.

SMYTH, H. F., JR., and SEATON, J. (1940): Acute

response of guinea pigs and rats to inhalation of the vapors of tetraethyl orthosilicate. *J. Ind. Hyg. & Tox.*, 22:288-296.

SMYTH, H. F., JR.; SEATON, J., and FISCHER, L. (1942): Response of guinea pigs and rats to repeated inhalation of vapors of mesityl oxide and isophorone. *J. Ind. Hyg. & Tox.*, 24:46-50.

SMYTH, H. F., JR., and CARPENTER, C. P. (1945). Note upon the toxicity of ethylene chlorhydrin by skin absorption. *J. Ind. Hyg. & Tox.*, 27:93.

SMYTH, H. F., JR.; CARPENTER, C. P., and WEIL, C. S. (1951): Range-finding toxicity data, List IV. *Arch. Ind. Hyg. & Occup. Med.*, 4:119-122.

SPECHT, H. (1938): Acute response of guinea pigs to inhalation of methyl isobutyl ketone. U.S. Public Health Service, *Pub. Health Rpts.*, 53:292-300.

SPECHT, H.; MILLER, J. W.; VALAER, P. J., and SAYERS, R. R. (1940): Acute response of guinea pigs to the inhalation of ketone vapors. U.S. Public Health Service, *Natl. Inst. Health Bull.* 176.

SPENCER, H. C.; IRISH, D. D.; ADAMS, E. M., and ROWE, V. K. (1942): The response of laboratory animals to monomeric styrene. *J. Ind. Hyg. & Tox.*, 24:295-301.

SPENCER, H. C.; ROWE, V. K.; ADAMS, E. M., and IRISH, D. D. (1948): Toxicological studies on laboratory animals of certain alkyldinitrophenols used in agriculture. *J. Ind. Hyg. & Tox.*, 30:10-25.

SPENCER, H. C.; ROWE, V. K.; ADAMS, E. M.; MCCOLLISTER, D. D., and IRISH, D. D. (1951): Vapor toxicity of ethylene dichloride determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 4:482-493.

SPOLYAR, L. W.; KEPPLER, J. F., and PORTER, H. G. (1944): Cadmium poisoning in industry. *J. Ind. Hyg. & Tox.*, 26:232-240.

STEINBERG, H. H.; MASSARI, S. C.; MINER, A. C., and RINK, R. (1942): Industrial exposure to tellurium. *J. Ind. Hyg. Toxicol.*, 24:183-192.

STERNER, J. H. (1943): Determining margins of safety. *Ind. Med.*, 12:514-518.

STERNER, J. H.; OGLESBY, F. L., and ANDERSON, B. (1947): Quinone vapors and their harmful effects. *J. Ind. Hyg. & Tox.*, 29:60-84.

STERNER, J. H.; CROUCH, H. C.; BROCKMYRE, H. F., and CUSACK, M. (1949): A ten year study of butyl alcohol exposure. *AIHA Quart.*, 10:53-59.

STERNER, J. H. (1955): The experimental animal—man—in industrial hygiene. *AIHA Quart.*, 16:103-107.

STOKINGER, H. E. (1949): Toxicity following inhalation of fluorine and hydrogen fluoride. (In C. Voegtlin and Hodge, Pharmacology and Toxicology of Uranium Compounds). National Nuclear Energy Series, Div. VI, Vol. I, pp. 1021-56. McGraw-Hill, New York.

SUNDERMAN, F. W.; WEIDMAN, F. D., and BATSON, O. V. (1945): Studies of the effects of ammonium picrate on man and certain experimental animals. *J. Ind. Hyg. & Tox.*, 27:241-248.

SUNDERMAN, F. W., and KINCAID, J. F. (1954): Nickel poisoning. *J.A.M.A.*, 155:889-894.

SVIRBELY, J. L.; HIGHMAN, B.; ALFORD, W. C., and VON OETTINGEN, W. F. (1947): The toxicity and narcotic action of monochloromonobromomethane. *J. Ind. Hyg. & Tox.*, 29:382-389.

SVIRBELY, J. L. (1954a): Acute toxicity studies of decaborane and pentaborane by inhalation. *Arch. Ind. Hyg. & Occup. Med.*, 10:298-304.

SVIRBELY, J. L. (1954b): Subacute toxicity of decaborane and pentaborane vapors. *Arch. Ind. Hyg. & Occup. Med.*, 10:305-311.

SWENSSON, A.; HOLMQUIST, C. E., and LUNDGREN, K. D. (1955): Injury to the respiratory tract by isocyanates used in making lacquers. *Brit. J. Ind. Med.*, 12:50-53.

TABERSHAW, I. R.; FAHY, J. P., and SKINNER, J. B. (1944): Industrial exposure to butanol. *J. Ind. Hyg. & Tox.*, 26:328-330.

TOUSEY, R. G. (1954): Malathion—Cyanamid's versatile insecticide. *Agr. Chem.*, 9:No. 7, 49-50.

TREON, J. F.; CRUTCHFIELD, W. E., and KITZMILLER, K. V. (1943): The physiological response of animals to cyclohexane, methylcyclohexane and certain derivatives of these compounds. *J. Ind. Hyg. & Tox.*, 25:323-347.

TREON, J. F.; SIGMON, H.; WRIGHT, H., and KITZMILLER, K. V. (1949): The toxicity of methyl and ethyl acrylate. *J. Ind. Hyg. & Tox.*, 31:317-26.

TREON, J. F., and CLEVELAND, F. P. (1955): Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. *J. Agr. Food Chem.*, 3:402-408.

TREON, J. F.; CLEVELAND, F. P.; CAPPEL, J., and ATCHLEY, R. W. (1956): The toxicity of the vapor of Arochlor 1242 and of Arochlor 1254. Paper read April 26, 1956 before the American Industrial Hygiene Association, Philadelphia.

U.S. DEPT. LABOR (1941): Control of welding hazards in defense industries. Division Labor Standards, *Spec. Bull. No. 5.*

U.S.P. H.S. (1943): Manual of industrial hygiene, W. M. Gafafer, editor. Saunders, Philadelphia, p. 264.

VIGLIANI, E. C., and ZURLO, N. (1955): Experiences of the Clinica del Lavoro with maximum allowable concentrations of industrial poisons. *Arch. Gewerbepath. u. Gewerbehyg.*, 13:528-535. (Abstracted in *Arch. Ind. Health*, 13:403, 1956.)

VON OETTINGEN, W. F.; HUEPER, W. C.; DEICHMANN-GRUEBLER, W., and WILEY, F. H. (1936): 2-Chlorobutadiene: Its toxicity and pathology and mechanism of action. *J. Ind. Hyg. & Tox.*, 18:240-270.

VON OETTINGEN, W. F. (1940): Toxicity and potential dangers of aliphatic and aromatic hydrocarbons. U. S. Public Health Service, *Pub. Health Bull. 255.*

VON OETTINGEN, W. F. (1941): The aromatic amino and nitro compounds, their toxicity and potential dangers. U. S. Public Health Service, *Pub. Health Bull. 271.*

VON OETTINGEN, W. F.; NEAL, P. A., and DONAHUE, D. D. (1942): The toxicity and potential dangers of toluene. *J.A.M.A.*, 118:579-584.

VON OETTINGEN, W. F. (1943): The aliphatic alcohols: their toxicity and potential dangers in relation to their chemical constitution and their fate in metabolism. U. S. Public Health Service, *Pub. Health Bull. 281.*

VON OETTINGEN, W. F.; DONAHUE, D. D.; SNYDER, R. K.; HORECKER, B. L.; MONACO, A. R.; LAWTON, A. H.; SWEENEY, T. R.; NEAL, P. A. (1944): Experimental studies on the toxicity and potential dangers of trinitrotoluene. U. S. Public Health Service, *Pub. Health Bull. 285.*

WAITE, C. P., and YANT, W. P. (1928): Microscopic pathology attending exposure of guinea pigs to vapors of ethyl bromide. U. S. Public Health Service, *Pub. Health Rpts.*, 43:2276-2282.

WAITE, C. P.; PATTY, F. A., and YANT, W. P. (1930): Acute response of guinea pigs to vapors of some new commercial organic compounds. IV. Ethylene oxide. U. S. Public Health Service, *Pub. Health Rpts.*, 45:1832-1843.

WATROUS, R. M. (1942): Methyl bromide, local and mild systemic toxic effects. *Ind. Med.*, 11:575-579.

WATROUS, R. M., and MCCAUGHEY, M. B. (1945): Occupational exposure to arsenic. *Ind. Med.*, 14:639-646.

WEAVER, F. L., JR.; HOUGH, A. R.; HIGHMAN, B., and FAIRHALL, L. T. (1951): The toxicity of methylal. *Brit. J. Med.*, 8:279-283.

WEBSTER, S. H. (1946): Volatile hydrides of toxicological importance. *J. Ind. Hyg. & Tox.*, 28:167-182.

WERNER, H. W.; MITCHELL, J. L.; MILLER, J. W., and VON OETTINGEN, W. F. (1943a): The acute toxicity of vapors of several monoalkyl ethers of ethylene glycol. *J. Ind. Hyg. & Tox.*, 25:157-163.

WERNER, H. W.; MITCHELL, J. L.; MILLER, J. W., and VON OETTINGEN, W. F. (1943b): Effects of repeated ex-

posure of dogs to monoalkyl ethylene glycol ether vapors. *J. Ind. Hyg. & Tox.*, 25:409-414.

WERNER, H. W.; NAWROCKI, C. Z.; MITCHELL, J. L.; MILLER, J. W., and VON OETTINGEN, W. F. (1943): Effects of repeated exposures of rats to vapors of monoalkyl ethylene glycol ethers. *J. Ind. Hyg. & Tox.*, 25:374-379.

WILEY, F. H.; HUEPER, W. C., and VON OETTINGEN, W. F. (1936): On the toxic effects of low concentration of carbon disulfide. *J. Ind. Hyg. & Tox.*, 18:733-740.

WILSON, R. H., and DEEDS, F. (1936): Chronic nicotine toxicity. *J. Ind. Hyg. & Tox.*, 18:553-564.

WILSON, R. H. (1944): Health hazards encountered in manufacture of synthetic rubber. *J.A.M.A.*, 124:701-703.

WINSLOW, C-E. A. (1927): Summary of the National Safety Council study of benzene poisoning. *J. Ind. Hyg.*, 9:61-74.

YANT, W. P.; SCHRENK, H. H.; PATTY, F. A., and SAYERS, R. R. (1930): Acrolein as a warning agent for detecting leakage of methyl chloride from refrigerators. U. S. Bur. Mines Rpts., Investigations 3027.

YANT, W. P.; SCHRENK, H. H.; WAITE, C. P., and PATTY, F. A. (1930): Acute response of guinea pigs to vapors of some new commercial organic compounds. II—Ethyl benzene. U. S Public Health Service, *Pub. Health Rpts.*, 45:1241-1250.

YANT, W. P.; SCHRENK, H. H.; and PATTY, F. A. (1932): The toxicity of dichlorotetrafluorethane. U. S. Bur. Mines Rpts., Investigations 3185.

YANT, W. P.; PATTY F. A., and SCHRENK, H. H. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. IX—Pentanone (methyl propyl ketone). U. S. Public Health Service, *Pub. Health Rpts.*, 51:392-399.

Reprints Available

REPRINTS of the preceding Cummings Memorial Lecture may be obtained from GEORGE D. CLAYTON, Executive Secretary, AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, 14125 Prevost, Detroit 27, Michigan, at $1.50 each. The price of this issue of the AIHA QUARTERLY is $2.00.

Dr. Henry Field Smyth, Jr., receiving the Cummings Memorial Award from N. V. Hendricks, President of the American Industrial Hygiene Association. The presentation was made at the banquet of the Association's Seventeenth Annual meeting in Philadelphia, Wednesday, April 25.

A DIRECT METHOD FOR THE

Collection and Determination of Micro Amounts of Benzene or Toluene in Air

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A SIMPLE, rapid and direct procedure for the collection and determination of micro quantities of pure benzene and toluene vapor in air is described. The sampling apparatus is compact, and portable, and the air samples may be shipped and stored without loss. The scope of the method is illustrated by a series of determinations with known quantities of benzene or toluene ranging from 0.5 mg to 5.0 mg. The analysis of a sample is completed in less than 30 minutes, and the aromatic hydrocarbon content of the sample is read directly from a graph.

The method is based on the optical absorption, at selected ultra-violet wavelengths, of benzene or toluene in iso-octane. To simplify the following description, only benzene will be mentioned specifically, but the procedure has been worked out for both hydrocarbons. Existing ultraviolet methods for benzene are too mathematical,[1,2] or only applicable to liquid samples.[2,3] By restricting the application of the method to exposures involving a single hydrocarbon, solution by simultaneous equations is eliminated.

The aromatic is collected by drawing a known volume of contaminated air through a sampling tube containing silica gel. The gel on which the sample is adsorbed is immersed in iso-octane (2,2,4-trimethylpentane); on addition of water, the hydrocarbon is displaced from the gel and dissolves in the iso-octane. The absorbance of this solution in the ultra-violet is measured on a spectrophotometer. The concentration of benzene can then be read directly from a calibration curve of absorbance versus concentration plotted previously from known samples.

Presented at the Seventeenth Annual Meeting of the American Industrial Hygiene Association, Philadelphia, April 26, 1956.

Apparatus and Reagents

THE absorbances are determined with a Beckman DU spectrophotometer with photomultiplier attachment (optional) using 5 cm cylindrical cells. Known amounts of benzene are measured with a Gilmont microburet. To prepare spectro grade iso-octane, technical grade iso-octane is passed consecutively through two columns of silica gel (Davison 22-200 mesh), 2½ inches in diameter and 11 inches in depth. The trans-

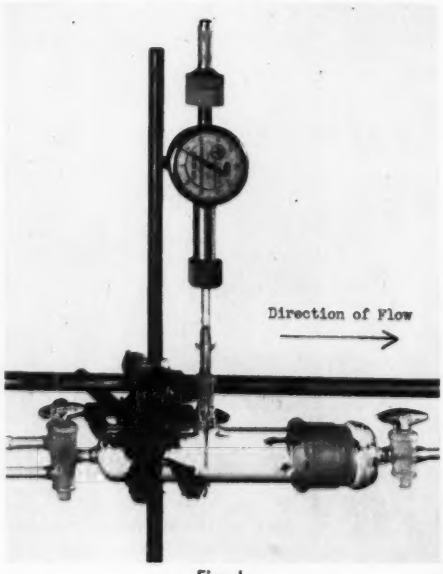

Fig. 1.

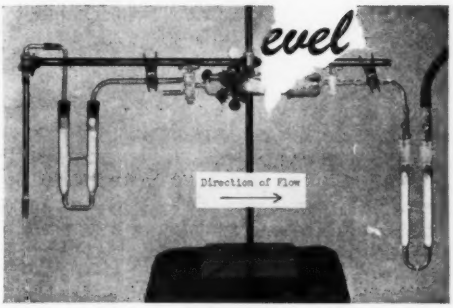

Fig. 2.

mittance of the purified material, compared with triple distilled water, was greater than 95% at wavelengths above 250 millimicrons. The most satisfactory silica gels for use in the sampling tubes are refrigeration grades PA 100 and PA 400 manufactured by Davison Chemical Company, Baltimore, and 14-28 mesh material from the Eagle Chemical Company, Incorporated, Joliet, Illinois. These gels come as free flowing granules, free from fine particles and dust. Other gels investigated broke up into fine particles on shaking, or contained substances which lowered the transmittance of purified isooctane.

Benzene and toluene used for standardization are N.B.S. certified materials from the Phillips Petroleum Company, Bartlesville, Oklahoma. The sampling tubes are similar to those used by Fahy to collect chlorinated hydrocarbons.[4]

Standardization Procedure

USING the microburet, a measured amount of benzene is placed on glass wool in a micro gas chamber (Figs. 1 and 2). Air is drawn through in the direction shown at the rate of 1200-1500 cc/min for five minutes. The benzene is adsorbed on 15 cc of silica gel in the first limb of the sampling tube. The gel is placed in a clean, dry, glass stoppered erlenmeyer flask to which is added 25 cc of isooctane and 6 cc of water. The mixture is shaken by hand for three minutes, and then allowed to stand for at least 10 minutes. The absorbance of the decanted solution is measured on the spectrophotometer against an iso-octane blank at sensitivity 2, and slit width 0.7 mm. Benzene solutions are read at wavelength 254.5 millimicrons and toluene solutions at 268.0 millimicrons. A calibration curve is drawn plotting absorbances against the weight of hydrocarbon taken (Table I). The amount of pure hydrocarbon in an air sample may be read directly from such a graph (Fig. 3).

Discussion

THE SAMPLES of benzene and toluene were completely adsorbed on the first portion of silica gel.

No measurable amount was ever de-

TABLE I.

Weight	No. of det.	Mean Absorbance	Range-weight found	Deviation
		Benzene		
0.25 mg	5	0.210	0.21 → 0.28 mgms	±17%
0.50	5	0.311	0.42 → 0.54	16
1.0	5	0.511	0.88 → 1.04	12
2.0	5	0.918	1.92 → 2.09	5
3.0	5	1.297	2.94 → 3.08	3
4.0	5	1.624	3.94 → 4.10	3
5.0	5	1.899	4.98 → 5.02	4
		Toluene		
0.20	5	0.161	0.15 → 0.22	±25%
0.50	5	0.276	0.42 → 0.56	16
1.0	5	0.446	0.92 → 1.06	8
2.0	5	0.811	1.96 → 2.04	2
3.0	5	1.10	2.90 → 3.10	3
4.0	5	1.38	3.84 → 4.14	4
5.0	5	1.57	4.74 → 5.16	5

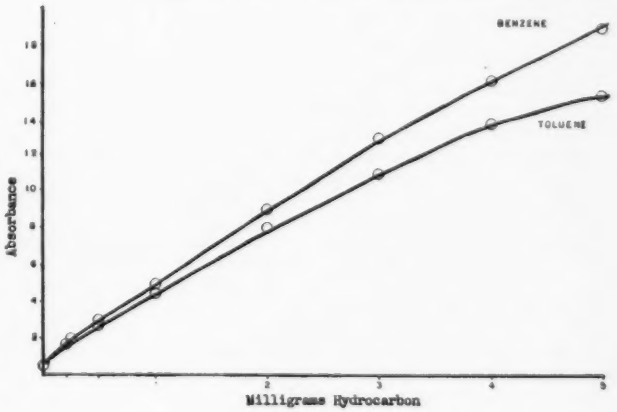

Fig. 3.

tected on the gel in the second limb of the sampling tube.

All the hydrocarbon was transferred from chamber to adsorber in five minutes. The percent recovery was not affected by lengthening the sampling time, indicating that there is no tendency for the hydrocarbon to be desorbed by prolonged air flow. A silica gel filter was placed upstream from the air chamber to prevent possible contamination by the laboratory air.

Absorbance-concentration curves, with identical slopes, were obtained by dissolving similar amounts of benzene in iso-octane.

The 5 cm light path chosen provides a reasonably steep curve, which makes it possible to determine the absolute amount of the aromatic to a high degree of sensitivity. It is possible to increase the sensitivity further by using the photomultiplier at a higher sensitivity, and by using capillary cells with the same light path which permits the use of less iso-octane for extraction. However, since the "Threshold Limit Value" for benzene (35 ppm), or toluene (200 ppm), can be detected by sampling at the rate of one cubic foot of air per minute for one minute, higher sensitivity hardly seems necessary. In fact, experience with air sa[illegible]es that a spectrophotometer [illegible] silica cells, and without a photomu[illegible] attachment, is perfectly satisfactory.

The use of w[illegible]er to extract the hydrocarbon from the gel is essential. The hydrocarbons cannot be removed from the gel unless water is added. We recognize the limiting circumstance that for results to be valid, sampling and analysis by this method can only be used where the occupational exposure involves a single aromatic hydrocarbon. As this is a common industrial situation it does not too seriously limit the application of the method.

Speed and simplicity of analysis, together with sensitivity and accuracy, make this a great improvement over other ultraviolet methods, and methods involving nitration.

References

1. GOLDMAN, F. H.: Laboratory Procedures in Industrial Hygiene. U. S. Public Health Service, 1947.

2. Tentative Method of Test for Benzene and Toluene by Ultra-violet Spectrophotometry. Proceedings, *Am. Soc. Testing Mats.*, 48:374, 1948.

3. TUNNICLIFF, D. D.; BRATTAIN, R. ROBERT; ZUMWALT, L. R.: Benzene, toluene, ethylbenzene, o-xylene, m-xylene, and p-xylene, Determination by Ultra-Violet Spectrophotometry. *Anal. Chem.*, 21:890-4, 1949.

4. FAHY, J. P.: Determination of Chlorinated Hydrocarbons in Air. *J. Ind. Hyg. & Tox.*, 30:205-7, 1948.

Department of One Paragraph Reports

HOUSEOWNERS are often faced with the necessity of removing old paint, frequently many layers thick, prior to proper re-decorating. In the instance reported here, the wooden baseboards, moldings, and window casings in a bathroom were concerned. A commercial paint remover of local manufacture containing carbon tetrachloride and trichloroethylene was used and warnings concerning adequate ventilation were observed by working with the one window open. The house is heated by oil-fired forced-draft hot air, with no return air duct in the bathroom. A strong wind directly into the bathroom from the open window was sufficient to virtually overcome the forced-draft hot air and actually blow vapors down into the heating dome of the furnace when the fan was on, and to more readily follow the same path with virtual complete distribution throughout the house when the fan was not operating. Shortly, the odor of the chlorinated hydrocarbons was acutely evident, and when working behind the closed door, the superimposed odor of phosgene was strongly apparent. A multiple toxic exposure thus occurred, the symptoms of which included headache, nausea, irritation to mucous tissues, and extreme nervousness. The headache, nausea and irritation departed on exposure to fresh air but the nervous symptoms continued for about three days. These included rapid and fluctuating pulse with palpitations, tremor of the hands, barely legible handwriting with tendency toward up-hill writing, and a halting gait. Medication with 0.5 grain of phenobarbital every four to six hours was without effect. While equipment for sampling was not available, it is suggested that the syndrome is the synergistic effect of carbon tetrachloride, trichloroethylene, and phosgene which resulted from thermal decomposition of the carbon tetrachloride on the hot dome of the furnace. —LYNN D. WILSON, Ph.D.

Ground Level Contamination

FROM STACK EFFLUENTS

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FACILITIES for the handling and processing of tonnage quantities of uraniferous materials have been in continuous operation since 1942. Many of these production plants, constructed during the emergencies and pressures of World War II, were not intended as permanent installations and therefore were built with inadequate dust collection devices.

After 1946 the Atomic Energy Commission restudied all plant operations with special emphasis on the efficiency of health, safety and production. A broad program of improving dust and fume control was instituted during the period 1948-50.

During the early years, uranium-bearing materials in substantial quantity were discharged to the atmosphere at various plant sites. This study has been directed toward an assessment of the effect of plant effluents fallout on the surrounding soil. It is believed that a study of this type might yield data which are of general interest and applicability.

Purpose

IN DETERMINING the long term accumulation of uranium in soils surrounding a plant a study was made at three A.E.C. processing sites in order to: (1) determine the concentration gradient of uranium residing in the soil with respect to depth, distance, and direction from the source of pollution, and (2) compare the results found at each plant.

Method of Study

SOIL SAMPLES were taken around each plant as follows:

1. A series of four concentric circles, of 500-foot, 1000-foot, 2000-foot and 4000-foot radii were circumscribed around each plant mid point. No samples could be obtained inside of 500 feet because most of this surface, being inside the plant fence, was paved.

2. Eight equally spaced radii were superimposed on the circles; the positioning of the radii was at the cardinal compass points.

Soil sample surveys were conducted at three A.E.C. facilities to determine the concentration gradients of uranium in the soil with respect to depth, distance, and direction from the source of pollution. These plants have been in operation for over 14 years and have discharged 18.5, 50, and 52 tons respectively of uranium as metal in the stack effluents. About 40% by weight of the effluent was estimated to be still in the ground at the two plants where the discharge was largely insoluble material. The soluble salts remained only to the extent of about 1.2%. The maximum average concentration of uranium was about 50 μg/gm at the 500 foot radius. There was no significant effect of direction from the source (Radius Effect). The soil concentration in each case decreased linearly with distance and was within the range of normal background of uranium in soil (3 - 9 μg/g) beyond about 2000 feet from the center of plant activities. Plants A and B showed a soil concentration decrease approximately as the square of the distance from the source. The top inch of soil contained about two to three times the concentration of the lower 5" where the material was insoluble, while no difference was found where the effluent was in the form of soluble salts.

TABLE I.
RANGE OF FALLOUT RESULTS

Distance from Source Feet	Type Samples		Plant A		Plant B		Plant C	
			Uranium in Soil Concentrations (μg/g)					
			Min.	Max.	Min.	Max.	Min.	Max.
500	Upper	Individual	4.6	390	5	65	1	29.5
		Average of Duplicates	10.3	298	7.25	53	2.25	21.5
	Lower	Individual	0.5	80	1.3	41	1.5	46
		Average of Duplicates	2.25	45	2.45	41	1.75	35
	Overall Average		46.9		36.0		10.3	
4000	Upper	Individual	0.15	1.5	0.9	4.9	0.1	6.5
		Average of Duplicates	0.15	0.875	1.475	4.5	0.25	5.5
	Lower	Individual	0.1	0.6	1.15	4.7	0.15	4.2
		Average of Duplicates	0.15	0.45	1.45	3.15	0.175	3.9
			0.33		2.4		1.16	

3. Where each radius intersected a circle, duplicate soil samples of the surface (0" to 1") and subsurface (1" to 6") were obtained by conventional soil sampling techniques.

Each sample was analysed photo-fluorimetrically for uranium content.

Production History and Climatological Data

PLANT A was engaged in drying, crushing, grinding, weighing, sampling, and packaging ore. The plant had a single effluent stack. The operating period was December, 1947, to December 1954. Local climatological data are: average annual temperature 54°F, average annual rainfall 45 inches, mean hourly wind speed 10 mph, prevailing direction south west.

The plant discharge averaged 8 x 10^5 $\mu g/M^3$ of uranium as determined by hourly samples taken one day each month for about two years. The air flow rate was 100 M^3/min for about 35 hours per week over the period. A computation on the basis of the total number of lots handled shows that about 50 tons of metal was discharged to the atmosphere over the seven years.

Plant B was several buildings with many individual stacks. The operation was the chemical processing of uranium ores to metal. Production operations have been continuous from 1942 to date. Local climatological data indicate a mean annual temperature of 57°F, a mean annual rainfall of 38 inches, average hourly windspeed of 11 mph, prevailing direction south.

TABLE II.
COMPARISON OF SURVEY RESULTS

Item	Plant A	Plant B	Plant C
Years of Operation	7	13	11
Average Annual Rainfall ("/yr)	45	38	38
Estimated Stack Discharge (tons)	47-55	18.5	52.3
Area Sampled (mi^2)	2	2	2
Soil Content Tons (per inch) Upper Soil (0" - 1")	4.3	3.0	0.1
Soil Content Tons (per inch) Lower Soil (1" - 6")	2.3	1.0	0.1
Soil Content Tons (per inch) Total (0" - 6")	16	8	0.6
Tons of Uranium in Soil per Ton of Stack Effluent	0.32	0.40	0.012

From extensive air sampling data obtained over many years from each individual stack, it has been estimated that a composite of 18.5 tons of metal was discharged as stack effluent.

Plant C was a single building with multiple stacks, engaged in converting UO_2 into UF_4 during the period 1942-1949. From 1944 for seven years, an additional step involving the conversion of UF_4 to UF_6 was added. By far the major share of the plant effluent came from this step as a hydrolysis product of gaseous UF_6 (probably UO_2F_2). From 1951 to December, 1952, the plant was involved exclusively in converting U_3O_8 to UO_3.

Local climatological data reveals an average annual temperature of 51°F, average rainfall of 38 inches, mean hourly windspeed 13 mph, prevailing direction south.

Continuous sampling of the main dis-

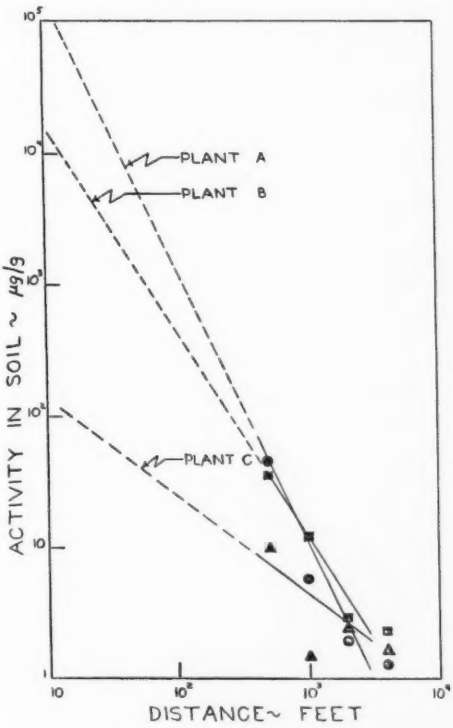

Fig. 1.
Average concentration of uranium in soil.

charge stacks with spot samples taken at each of the other discharges indicated that approximately 52 tons of uranium was discharged to the atmosphere during the life of the operation.

Quantitative Analysis of Results

TABLE I is a compilation of maximum and minimum concentrations of uranium in soil obtained at the plant sites. Table II summarizes the data which are plotted in Fig. 1. A somewhat hazardous extrapolation has been made to cover the unsampled area from 10 to 500 feet in order that an estimate could be made of the theoretical total quantity in the soil. It should be noted that beyond about 2000 feet the results are not distinguishable from normal uranium background. Analysis of these reveals the following:

1. PLANT A: Of the total amount of uranium discharged in the stack effluent, 32% by weight (16 tons) was estimated to be in the two square miles sampled. The maximum average concentration found was 46.9 micro grams/gram at 500 feet. The concentration declined approximately as the square of the distance. The maximum individual sample of 390 μ grams per gram was obtained in the upper soil at 500 feet. At 4000 feet the range was 0.1 to 1.5 μg/g. This is undoubtedly background.

2. PLANT B: Approximately 40% (7.9 tons) of the uranium discharged in the stack effluent was found in the two square miles of sampled land area. The average concentration was a maximum of 36.0 μ grams/gram at 500 feet. The maximum concentration (65 μg/g was found in the upper soil 500 feet from the source. At 4000 feet no concentration of uranium in soil exceeded 4.9 μg/g.

3. PLANT C: Approximately 1.2% (0.6 tons) of the uranium discharged in the stack effluent was found in the sampled land area. The maximum average concentration of uranium in soil was 10.3 μg/g at 500 feet. The maximum concentration for an individual sample was 46 μ grams of uranium per gram of soil. This was obtained in the lower soil at the 500-foot radius. At 4000 feet, which was the maximum distance from the source for sampling, no concentration exceeded 6.5 μg/g.

Discussion

A STATISTICAL analysis (Table III) shows that there was no significant effect of direction from the source (Radius Effect) on soil concentration in any case. On the other hand, the fall-off of concentration with distance (Circle Effect) was highly signifi-

TABLE III.
STATISTICAL ANALYSIS OF RESULTS

Source of significance	Plant A	Plant B	Plant C
Radius			
Circle-Linear	H.S.	H.S.	H.S.
Circle-Quadratic		H.S.	S.
Circle-Cubic			
Sample			
Radius x Circle			S.
Radius x Sample			
Sample x Circle-Linear			
Sample x Circle-Quadratic			
Sample x Circle-Cubic			

NOTE:
S. = Significant
H.S. = Highly Significant
All Blanks = Not Significant

TABLE IV.
EXTRAPOLATED QUANTITIES—SUMMARY

Annulus (Feet R)	Area sq. ft.	Plant A Ave ug/g	Plant A Total Tons	Plant B Ave ug/g	Plant B Total Tons	Plant C Ave ug/g	Plant C Total Tons
10-40	4.7×10^3	3.5×10^4	5.6	9×10^3	1.6	85	0.04
40-100	26×10^3	3.5×10^3	4.0	1.3×10^3	1.2	40	0.04
100-200	10^5	650	2.4	330	1.2	20	0.04
200-400	3.7×10^5	135	2.0	100	1.4	10	0.12
400-1000	2.6×10^6	17.5	1.6	17.5	1.6	2.5	0.24
1000-2000	9×10^6	1.0	0.4	3.0	0.8	0.5	0.12
2000	—	Bkgd*		Bkgd*		Bkgd*	
TOTAL TONS			16		7.9		0.6

*Background assumed to be 2.5 ug/g.

cant in all three cases. Within the limits of experimental error, the decrease of concentration occurred approximately as the second power of the distance for plants A and B. Plant C showed a much flatter decrease.

In view of the strong linear relationship of the average concentrations with distance, an attempt was made to estimate the total quantity that might have been found had the area been sampled back to zero distance. The three curves were, therefore, extrapolated in each case to 10 feet from the source, and the total quantities integrated. (Table IV) Interestingly enough, the material from both Plant A and Plant B was found to remain in the soil to the extent of between 30% to 40% of the estimated initial fallout. This would indicate that where highly insoluble materials are discharged, there is relatively small reduction in the soil content due to weathering. Also, most of the material appears to remain in the upper portion of the soil. About two to three times as much was found in the first inch as was in the next five. Plant C which discharged almost entirely a soluble form of uranium (UO_2F_2) showed a markedly different effect. Here the residual concentration in the soil was estimated to be approximately 1.2%. Similarly, the soluble material, differed in that it showed no significant difference between the concentration in the upper inch and the next five inches. The consistency between the results obtained at the three plants is remarkably good when the effluent conditions are taken into consideration.

Appendix

CRITERIA FOR SOIL CONTAMINATION: According to Table III, National Bureau of Standards Handbook No. 52: (1) The maximum permissible amount of soluble natural uranium in water for continuous exposure is 7×10^{-5} microcuries uranium per milliliter of water. This is equivalent to 110 micrograms of uranium per gram of water (ug/gm). This should be a conservative maximum for uranium soil contamination. (2) The natural soil uranium as reported by D. E. Lynch* is three to nine ug/g.

*LYNCH, D. E.: Soil and Water Uranium and Radium Survey. U.S.A.E.C., NYOO No. 1521, 1950.

I.L.O. Booklet Available

A PANEL of the Correspondence Committee on Occupational Safety and Health of the International Labour Office met in Geneva, Switzerland in December of 1954. The purpose of the meeting was to draw up guiding principles for the organization of occupational medical services. The Committee has prepared a 10-page document setting forth such principles. Copies of the booklet are available at 15c each from the Washington office of the I.L.O. Inquiries and orders should be addressed to R. PETER STRAUS, 917 15th Street, N. W., Washington 5, D. C.

PROBLEMS IN THE CALIBRATION OF

Industrial Hygiene Instruments

ELGIN D. SALLEE and ROBERT H. MILLER
Health and Safety Division, American Can Company, Newark

OPERATIONS of most industrial hygiene units are such that field test instruments are subjected to rather harsh treatment during transportation and subsequent use in the plant. The instrument must incorporate a practical combination of (1) unusually sensitive and accurate mechanism, since the apparatus is employed for measurement of micro quantities, (2) ruggedness to withstand the treatment received, (3) convenient portability, (4) rapidity in obtaining test results, and (5) convenience of operation. Ideal combinations of these requisites in present day instruments are comparatively rare. In addition, new materials and manufacturing processes are being developed daily, necessitating frequent revisions in types and uses of hazard-evaluating equipment. It is common practice to extend usefulness of certain instruments by employing them for evaluation of conditions or materials other than those for which they were designed primarily. As a result, proper calibration and frequent checking to assure desired operating efficiency of the instruments is recognized as a major function in industrial hygiene work.

It is particularly disconcerting to first note that an instrument is not operating properly, or that there is reason to doubt its accuracy, when the hygienist is already at the plant location where tests are to be made. It is much worse, of course, if the inaccuracies go undetected. Checking of the instrument in the field is equally as important as the sampling or evaluation test itself. Proper calibration beforehand for the intended use is imperative. The ideal instrument would be one that can be checked for accuracy and proper calibration conveniently at the point of use. The reliable instrument manufacturer ordinarily incorporates equipment and directions for a few simple check tests as an integral part of the operation of the apparatus. Importance of following these directions during field use cannot be overemphasized. In most cases, however, actual calibration of the apparatus must be done by the instrument manufacturer or in the industrial hygiene laboratory.

Specific problems are encountered in the proper calibration of practically all industrial hygiene instruments. Calibration work on apparatus for evaluation of air pollution, radiation and noise are particularly critical. Time allotted does not permit discussion of these problems since each is a subject in itself. Consequently, this paper is confined to only two types of instruments which have been in common use for a number of years by the majority of industrial hygiene units.

Solvent Vapor Determination

ACCURATE determination of solvent vapor concentrations in workroom air continues to be a major problem to industrial hygienists. The problem is complicated by the fact that there are so many types of solvents and so many solvent mixtures employed in industry. It is not uncommon to find six or more different solvents in a single organic coating material. There are thousands of different solvent mixtures in use, under varying conditions of temperature, pressure, handling methods, etc., which further complicate convenient vapor concentration measurement. Practical field use methods and instruments for evaluation of specific components of solvent vapor mixtures in workroom air are extremely rare. Consequently, in many cases it is expedient and practical to adapt available instruments and methods to meet the particular conditions and still assure workably accurate results. This necessitates instrument calibration. Our industrial hygiene unit prefers use of instruments which measure heat of com-

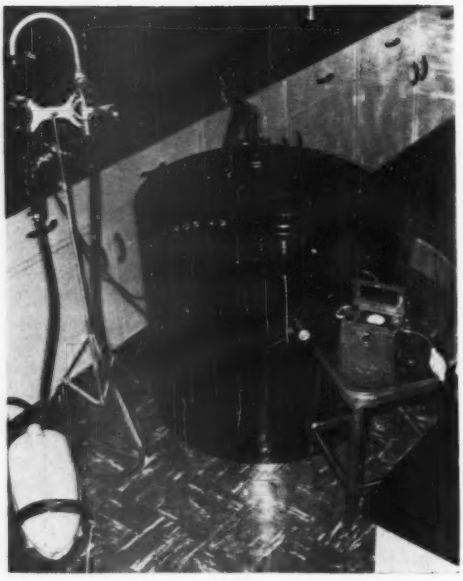

Fig. 1.
A 500-liter metal tank was used for calibration.

bustion for general solvent vapor evaluation work.

The common procedure for calibrating methods and apparatus for evaluation of solvent vapors involves sampling known vapor concentrations. Various methods have been suggested in the literature for producing known concentrations of vapor in air. Several of these have been tried in our laboratory but we still prefer the procedure whereby a known weight of solvent or solvent mixture is volatilized in a metal tank of known volume. In our laboratory we use a 500 liter tank (Fig. 1), fitted with an air mixing fan, a device for crushing glass ampoules containing the solvent, a heating element to volatilize solvents of low vapor pressure, a thermometer and various openings for sampling and removing and replacing the vapor-laden air. In use, the accurately weighed solvent, in such amount as to produce the approximate maximum vapor concentration desired, is placed in the tank and volatilized. The mixture is sampled and the results recorded. The concentration is then reduced, stepwise, by drawing out measured increments of the air-vapor mixture and replacing it with fresh air. The remaining known concentration mixture is sampled after each increment is withdrawn. The instrument readings are then plotted against the known concentrations sampled and a calibration curve is drawn. Typical calibration curves for various solvents and solvent mixtures are shown in Figs. 2, 3 and 4.

Experience has shown that the two instruments used for field work by our industrial hygienists can be employed interchangably, the same individual calibration curve applying to both instruments. Table I shows typical experimental data obtained in the comparison of calibrations for the two instruments by the aforementioned method. Readings are reproducible within $\pm$ 10%, as claimed by the manufacturer.

Experience has shown it advisable to check operation of the instruments prior to each major field trip. This includes a laboratory calibration check against a known concentration of solvent vapor and comparing the instrument reading with a previously determined calibration curve. For checking during field use, it is advantageous to equip the instrument so that it can be "zeroed" in a vapor-laden atmosphere. This involves attaching an activated charcoal chamber through which the intake air may be diverted, to remove the vapors, prior to being drawn through the instrument.

Further assurance of proper operation may be obtained conveniently in the field by checking the instrument against a known concentration of styrene. Apparatus for this consists merely of a quantity of styrene contained in a glass bottle with inlet and outlet tubes arranged so that air drawn

TABLE I.
COMPARISON OF SOLVENT CONCENTRATIONS EQUIVALENT TO A READING OF 0.3 (10-1 SCALE) ON TWO DIFFERENT M-6 VAPOTESTERS

	PPM Solvent at Reading of 0.3 (10-1 Scale)		
Solvent	M-6 Vapotester Serial No. 50-057-6	M-6 Vapotester Serial No. 670-047	Percent Difference Between Readings
Benzene	367	350	4.74
N-Butanol	773	740	4.16
Cyclohexane	393	369	6.11
Ethyl Acetate	816	741	9.19
Methanol	1296	1251	3.47
Methyl Isobutyl Ketone	621	609	1.93

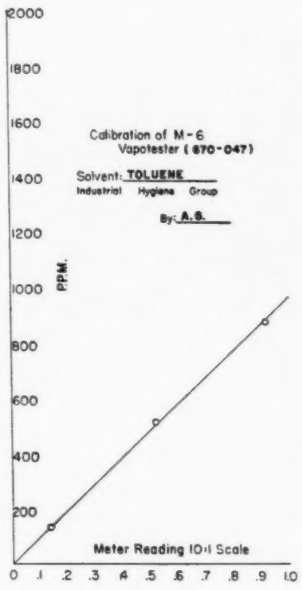

Fig. 2.

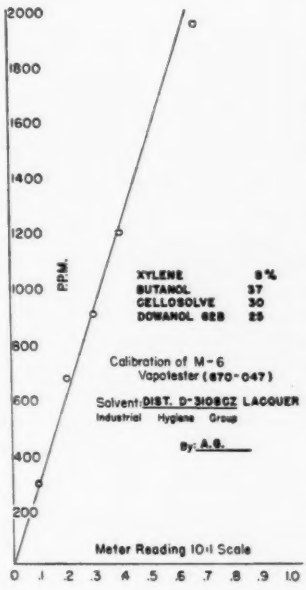

Fig. 3.

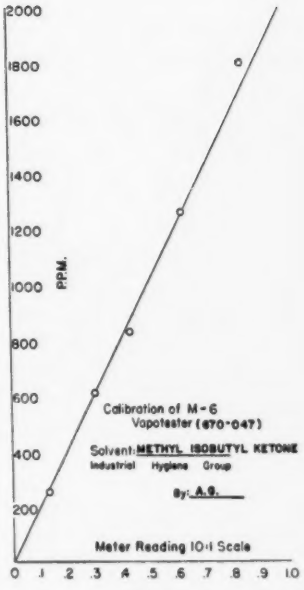

Fig. 4.

through the bottle contacts a large surface area of the styrene. The calibration test bottle is connected directly to the instrument and air is drawn through both by operating the aspirator bulb in the usual manner.

These instruments are employed for determination of all readily combustible vapors. Since they are not selective for any individual component of a vapor mixture, a reading is obtained on the "total combustibles" in the air and subsequently translated into vapor concentrations. It is necessary to assume that the proportions of individual components in the vapor are approximately the same as those existing in the liquid solvent mixture. A sample of the liquid mixture is obtained, simultaneously with the air testing, for subsequent calibration work in the laboratory. This consists of vacuum distillation at low temperature and subsequent analysis of the distillate by fractional distillation. The initial vacuum distillation results in separation of the mixture in proportions more closely approximating those actually existing in the workroom air. To assure adequate safety margin in exposure evaluation, maximum concentration limits are established for our specific Company use for each solvent mixture. These are based on the most toxic component of the mixture and are considerably below the A.C.G.I.H. recommended maximum allowable concentration values.

Electrostatic Dust and Fume Sampler

COLLECTION efficiency of this popular instrument has been proved through previous investigations reported in the literature. Our laboratory has never experienced difficulty in maintaining collection efficiency as long as the electrical system is adjusted and operated as directed. However, difficulty has been experienced in assuring accuracy of air-sample volume.

On older models of this apparatus an air flow-meter was provided. This could be checked periodically by means of the usual manometers, dry test meters, rotameters, etc. On the current, more convenient models, no gauge is provided since a constant speed motor and fan are incorporated in the "sampling head." Realizing the variation in air volumes handled by plant exhaust ventilation systems, most industrial hygienists are loathe to assume that the air vol-

TABLE II.
VALIDATION OF CO_2 METHOD FOR CALIBRATION OF ELECTROSTATIC PRECIPITATORS

Test Number	Volume Withdrawn - With Rotameter, C.F.M.	Calculated Volume Withdrawn-From CO_2 Concentrations, C.F.M.
1	3	3.06
2	3	3.07
3	2	1.84
4	2	2.11
5	2	2.25

TABLE III.
CALCULATED RATE OF AIR FLOW FOR ELECTROSTATIC PRECIPITATORS

Test Number	Electrostatic Precipitator Model No. 43184 Vol., C.F.M.	Model F Serial No. 878 Vol., C.F.M.	Model F Serial No. 906 Vol., C.F.M.
1	1.95	2.82	2.77
2	1.98	3.18	2.75
3	1.96	3.10	2.94
4	—	2.74	2.84

ume handled by current model samplers will remain constant. The problem in checking this is complicated by the fact that common air volume measuring apparatus cannot be employed since the added resistance of the flow meter would reduce the volume.

In checking this, therefore, our laboratory has found it practical to employ the 500 liter "calibration" tank mentioned previously. The procedure entails introducing a quantity of carbon dioxide into the tank, thorough mixing with the air in the tank at room temperature and pressure, and then measuring the carbon dioxide concentration by means of an Orsat apparatus. The electrostatic sampler head is then inserted into the tank and allowed to draw out an increment of the mixture, under its own power, for an accurately timed period. Fresh replacement air is drawn into the tank through an opened port so that there is no change in pressure and no change in air flow rate. Determination of carbon dioxide concentration in the remaining mixture is then made and the extent of reduction of carbon dioxide concentration related to the air flow volume. Volume rate is calculated by the following formula:

$$\text{Volume rate (in c.f.m.)} = \frac{V_t\ (\log C_i - \log C_f)}{0.431T}$$

in which V_t = Tank volume in cubic feet
T = Time interval in minutes
C_i = Initial CO_2 concentration
C_f = Final CO_2 concentration

Table II shows the data obtained in determining whether the procedure was feasible. In this instance a suction pump was used to withdraw the carbon dioxide laden air, with a rotameter for measuring airflow rate. Table III shows the results obtained on various checks of three separate electrostatic samplers. These data indicate the design volume rate has been maintained satisfactorily.

Summary

INSTRUMENTS used in industrial hygiene field work require frequent checking and calibration to assure continued accuracy. Usefulness of some instruments can be extended by employing them for measurements of materials or conditions other than those for which they were designed primarily, providing they are properly calibrated. Detailed procedure is given for calibration of instruments used for determination of solvent vapor concentrations. A method is described for checking sampling rate of present model electrostatic dust and fume samplers and other apparatus so constructed that checking by ordinary air-flow measurement equipment is not feasible.

Air Pollution Bibliography

A BIBLIOGRAPHY on air pollution will be prepared for the public health service by the Library of Congress. The bibliography will include references to the physical, biological, engineering, legal-administrative, and economic aspects of atmospheric pollution. This is the fourth in a series of agreements between the public health service and federal agencies for research in air pollution. The Bureau of Mines, the National Bureau of Standards, and the Weather Bureau are already conducting studies in air pollution for the Public Health Service.

Air Pollution Methodology

W. C. L. HEMEON, Director
Hemeon Associates, Pittsburgh

AN AIR POLLUTION study is soundly conceived and can provide significant results only if the methods are matched to the particular objectives of the undertaking. The objectives may be any one of the following: (a) to ascertain the cause of smog, as in Los Angeles; (b) establishment of levels of air pollution for geographic comparisons either within the community or between communities; (c) the establishment of levels for historical purposes and thus provide a basis for describing evolutionary changes; or (d) to ascertain the origin and relative importance of community dust nuisances.

In developing our subject, we have occasion to distinguish between the application of physical methods, e.g., measurement of soiling, dust fall, light-scattering indices, etc., and other types of measurements which identify elements or compounds, including analytical chemistry. It might be well to deal with the chemistry aspect first since it has often been our feeling that surveys sometimes become unduly preoccupied with the chemistry of air pollution whether it properly applies to the particular problem or not. The industrial hygienist when first projected into outdoor air pollution problems, is sometimes inclined, erroneously, to carry his concepts and in-plant techniques with him.

In industrial hygiene practice a quantitative survey is based on a study of a particular industrial process from which inferences are drawn concerning the character of contaminants and the air supply program planned for particular objectives. These objectives are either of two: to ascertain whether a hazard to worker health exists or whether concentrations of some non-toxic, though obnoxious, substance exceeds levels of decency as defined by its maximum allowable concentration. The chemical identity of the substance, as well as its concentration, is a fundamental requirement in industrial hygiene practice, for in the one case, health, this fact is related to the biochemical aspects of its toxicology and in the other case, worker comfort, the chemical identity leads to recognition of the objectionable quality. Unlike the situation applying to the in-plant problem, we have no criteria describing a relationship between outdoor atmospheric concentrations of some substance and a hazard to health and it is quite unlikely that we shall have such criteria in the foreseeable future (barring possibly some applicable to particular localities and of quite limited application).

There is sometimes observed in the design of a survey the apparent intent to characterize the atmosphere of a locality by detailed chemical analysis of as many substances as can be handled in the laboratory. Results of such work, however, disclose a monotonously similar character in the atmospheric solids in different cities throughout the country and such differences as are seen are discouragingly devoid of major significance.

There is some basis for the belief that the extensive scientific studies which have been conducted in Los Angeles during the past ten years have led to confusion in the minds of some as to methods of air pollution measurement that are generally applicable outside that region. It should be pointed out that the problem in Los Angeles is unique in several respects. The most important one here being that no one knew for many years what the cause of smog was and all technical research was devoted for a long period to answering this question.

This work led to a hypothesis concerned with chemical reactions involving ozone, oxides of nitrogen, and hydrocarbons the validity of which has been confirmed during the past year. The current research ac-

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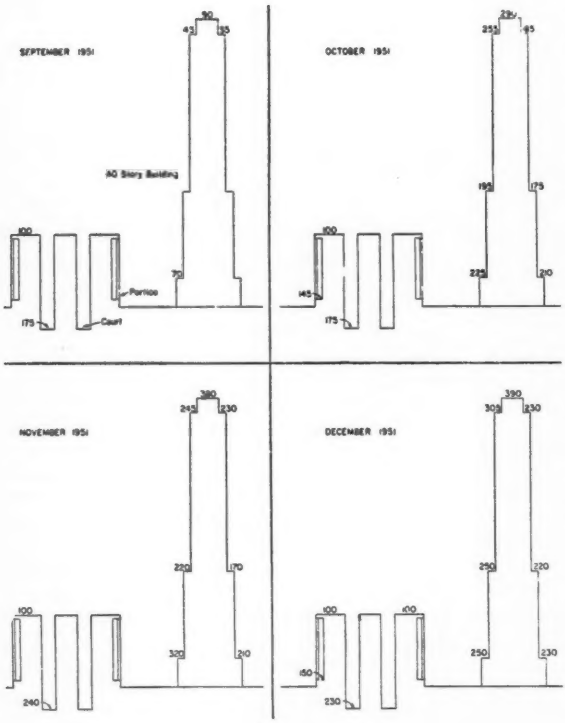

Fig. 1.

tivity in that area is now concerned with studies to evaluate the relative importance of the different gaseous ingredients and of the various potential sources. It is obvious then why measurements of air pollution in that area would be concerned mainly with measurements of these gases.

These measurements, however, are not generally applicable to the problems in other parts of the country unless one assumes an unrealistic attitude and prefers to concentrate on a study of the Los Angeles phenomena which may only possibly come to be of some future concern in other regions, at the expense of a study of presently existing problems that are mainly due to combustion of fuels.

Establishment of Air Pollution Levels

IF WE LOOK for common denominators in air pollution of our cities and industrial districts, we can name four primary air pollution qualities which embrace the largest segment of the problem generally. These are: (1) dust fall; (2) smoke; (3) the sulfur gases, sulfur dioxide and hydrogen sulfide; and (4) miscellaneous odors. We might, if we wish, create a fifth category to serve as a catch-all for miscellaneous pollutants having high significance in particular localities and fluorides would be one of those heading the list in this group.

It is interesting that we can describe two of these, dust and smoke, without reference to their chemistry at this stage of the discussion. This is important because it is related to the question of measurement methods and leads us immediately to select physical rather than chemical methods.

DUST FALL: Methods for measuring dust fall are well known. The cylindrical open-top vessel method in common use in this country was first used in Pittsburgh in 1912 as a part of the early smoke investigations at Mellon Institute. Sensenbaugh and Hemeon[1] studied the various factors that influence results and recommended a standard procedure which includes the following elements: (1) jars should be exposed at several locations to obviate the inherent error of a single jar; (2) they should be mounted at an elevation of two to four feet above the supported surface; (3) the jar bottom should be kept permanently wet.

Other possible sources of error in this measurement method are concerned with "stratification," a term employed here to indicate the high variability in dust fall intensity which often (though not always) prevails even within a small area. This is in sharp contrast to distribution of smoke concentrations as has been shown elsewhere.[2]

One illustration of this tendency to stratification is seen in the result of some measurements made[3] a few years ago at the location of our research headquarters. Dust fall jars were distributed at several locations and at different elevations within a small area including various elevations up

to the top of the 40-story tower of the University of Pittsburgh. The results of the measurements for a four month period are displayed in Fig. 1. We were confident that dustiness at high elevations would be materially lower than those near the ground and we were therefore completely surprised to find the reverse; dust fall rates on top of the tower were frequently four times those found on the lower roof top of our own building. This of course is to be explained in terms of local circumstances and it should not be concluded that the same would be true of another locality. These data do serve to emphasize, however, the importance of exploring the distribution of dust fall in a locality before characterizing the dust fall intensity.

SMOKE MEASUREMENTS: In recent years the measurement of smoke concentrations based on the intensity of stains produced by aspirating air through filter paper[4] has become common and is fundamentally logical. In a previous publication[5] we have stressed the importance of distinguishing between weight concentrations of air-borne solids and optical concentrations as exemplified by the COH unit. We have argued that one should not expect concordant results between two such methods of measurement since the weight of a few large particles may completely overbalance the mass of smaller particles. Recently comparative data due to some studies made by Cholak et al.[6] on the Los Angeles atmosphere have become available permitting direct comparison of the two methods of measurement, one employing the AISI automatic smoke sampler with concentrations expressed in COH units and other samples obtained by a high volume sampler from which a weight concentration is derived for the same location (see Fig. 2). At first glance, there appears to be good agreement between the two methods and indeed extreme upward and downward swings in concentrations are parallel to a great extent. However, we would call attention to, and emphasize the importance of the extent to which the two sets of data disagree. This would be even more apparent had we plotted the ratio between the two for each day.

A similar comparison also due to Cholak[6] is seen in Fig. 3 where the weight concentrations of lead in the atmosphere are compared with smoke concentrations but this time on an hourly basis. The same observations should be made. As expected, there is not a constant ratio between the two sets of results.

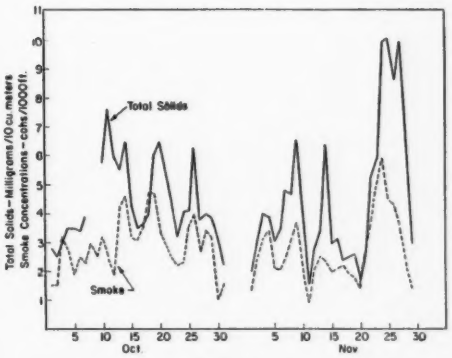

Fig. 2.
Comparative concentrations of solids on weight basis and on "COH" basis for Los Angeles—1954 (after Cholak).

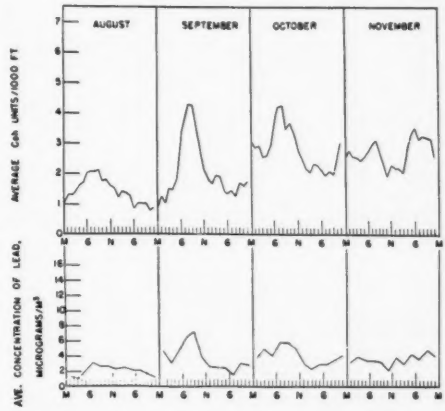

Fig. 3.
Fluctuations in the density of haze and in the concentration of lead averaged for each hour of the test period (after Cholak).

THE SULFUR GASES: Apparatus and procedures that are readily applicable for the determination of sulfur dioxide concentrations are well known. They are the Thomas autometer, the Titrilog, and the manual method, aspiration through alkali solution and iodine titration. Sensenbaugh and Hemeon[7] attempted the development of an

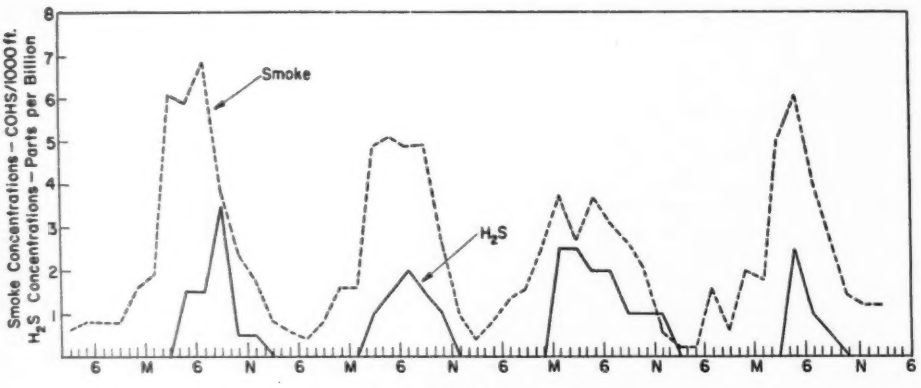

Fig. 4.
Hourly concentrations of smoke and of H_2S (Oakland Area of Pittsburgh) May, 1954.

automatic sulfur dioxide sampler involving adaptation of the AISI automatic smoke sampler using filter paper impregnated with a reagent that produced a sensitive color change on exposure to low concentrations of SO_2. This development was not completed.

The development of the hydrogen sulfide sampler involving the same system and filter paper impregnated with lead acetate makes it possible to measure concentrations of hydrogen sulfide somewhat lower than one part per billion parts of air and this instrument can also serve usefully in some air pollution surveys.

Single Air Pollution Index

THE EXTREME diurnal variation in concentrations of any air pollutant due to changes in atmospheric turbulence is well known. The question arises as to whether some one measurement such as that for sulfur dioxide can serve as a useful index of the air pollution intensity of a locality. In Fig. 4 we have plotted the comparative concentrations of hydrogen sulfide and of smoke at the same location for a period of several days from which it is apparent that concentrations vary in synchronism. These data are a small sample of more extensive data in our files and demonstrate that continuous measurements of any one pollutant can provide a useful index of the variations in concentration of all pollutants.

Measurement of smoke concentrations with the automatic filter paper sampler are the simplest of all measurements to make and therefore we prefer it for this purpose. Use of the data from a continuously operating automatic smoke sampler serves valuably for the evaluation of grab samples of some other constituent like sulfur dioxide which in the absence of such background data could not be properly averaged. In other words, a limited number of samples for some gas like sulfur dioxide can be tabulated to a significant average figure when background data from the smoke sampler is available.

Ascertaining Origin and Relative Importance

THE PRECEDING discussion applies to data obtained for historical purposes or for comparisons, as between localities or to different time periods.

In another phase of air pollution methodology one may be concerned with the problem of identifying sources of pollution and of assessing their relative importance. There are two primary approaches to such studies, one of them dynamic, involving application of micrometeorology, and the other static, applicable mainly to dusts.

MICROMETEOROLOGY STUDIES: Where instruments are available for hourly samples of significant air pollution qualities, useful information can be derived on the contribution of air pollution from different points of the compass when suitable meterological operations are made. The sampling instruments for smoke, sulfur dioxide, and hydrogen sulfide referred to previously provide

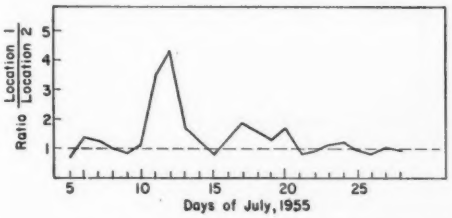

Fig. 5.
Comparative smoke concentration in two different localities in Pittsburgh.

the necessary tools for such studies. An hourly dust fall sampler is also needed for such studies and it was this need which impelled us to the development of such an instrument.[8] The directional dust fall sampler due to Munger was developed for similar objectives but since it must operate for a period of several days at least to acquire weighable samples, it is based on a procedural philosophy that is quite different from that implicit in those being discussed here.

In an application of the smoke sampler to such a study the data from three instruments operating in different locations simultaneously is best processed by calculating and plotting ratios of concentrations in order to cancel out the effects of diurnal variation due to changes in atmospheric stability. When neither of two samplers is subject to the influence of any directional source then the ratio in an ideal case would be unity and the resulting plot would be a horizontal, straight line. Local sources of pollution affecting one but not the other will show up as peaks and will direct attention to some particular wind direction or other weather factor. This is illustrated in Fig. 5.

STUDYING EMISSIONS FROM ONE STACK: During the past several years, a considerable amount of research has been devoted to studies on the rate of dispersion, of stack gases and the pollutants they carry in relation to stack design and various meteorological parameters, studies which involve the need for conventional weather instruments as well as one for measuring the elusive quality of turbulence. The latter is beyond the scope of this paper, but we may consider those aspects with which industrial hygienists may sometimes be confronted, namely the problem of sampling at ground level in the surrounding terrain and sampling of the effluents in the stack gas stream itself. Within the context of the present discussion, the instruments for dust fall, smoke, and the sulfur gases are appropriately applicable to studies involving these pollutants and in measurements of the pollutants in the stack gas stream similar measurements are applicable.

Everyone is now cognizant of the very special procedures and precautions necessary in the measurement of solids in stack gases. Not so well appreciated is the logic in measurement of smoke concentrations in the stack gas stream by filter paper sampling either manually or automatically, as a supplement to the conventional measurement of weight. If it is logical to distinguish between dust fall solids and smoke in the outdoor atmosphere, it is equally logical to make the same measurements in the stack gas stream. We have described elsewhere[4] the basis for the filter paper stain measurement and how the results can be evaluated and described in the same units of optical density as applies to ground level samples. Thus to fully describe a particular stack emission two separate sets of figures are employed, one describing the solids emission rate in weight units, e.g., grains per cubic foot or pounds per hour, and the other in units of optical density, *i.e.*, COH-ft.^{2} per minute.

TRACER TECHNIQUES: Where objectives warrant the effort, the contribution of a particular stack emission to the total of the surrounding region can be evaluated by tracer techniques that have been applied on an increasing scale in recent years. If a particular stack were emitting a pollutant having a unique chemical composition that is quite different from that of pollutants found in the surrounding atmosphere a direct study in analytical chemistry could serve the purpose of ascertaining the quantitative effect of this emission on the neighborhood. This is almost never the case, however, but the same results can be realized whereby a selected substance is injected into the stack gases at a measured rate and where samples are taken at ground level at varying distances and significant locations. The substance to be employed must, of course, be unique and determinable quantitatively with a high degree of sensitivity in order that the methods have maximum utility. One

tracer substance which has had extensive application is the one described by workers at Stanford University and subsequently applied by Stanford Research Institute in which a selected fluorescent powder is employed.[9a] We have also been conducting studies toward the development of alternative techniques for this same purpose.

DUST ANALYSIS: Allusion was made earlier to the limitations of chemical analysis in most air pollution studies. It can, however, have important application in examination of community dust samples for the purpose of judging the origin of various constituents and their importance. Conventional analysis, however, does not often lead to significant results because of the monotonous regularity with which one encounters the common elements, silica, iron, alumina, calcium, magnesium, carbon, and sulfur. Their proportions do vary but one is as likely to find variations in one locality from season to season as will be found between different studies and different locations within the city.

In an effort to develop laboratory examination procedures which would lead to more significant results, we in our laboratory have devoted considerable study to the problem with particular reference to settled dust. Simple methods have been devised[10] for separation of magnetic constituents from non-magnetic and for the separation of the latter portion into fractions of different specific gravity, utilizing a technique of sedimentation in liquids of high specific gravity. The separated fractions may be subjected to chemical analysis, to x-ray diffraction analysis, microscopic examination, and various other physical techniques. Such methods of dust "finger printing" can be much more illuminating than the conventional methods.

REFRACTIVE INDEX SPECTRUM: Near the end of this particular program of research, we conceived of a dust examination method which we believed to be "unique."[11] If a series of suspensions of the dust in question were made up with liquids of varying index of refraction and the optical density determined by transmission of light through the suspension, one might expect a significant increase in the quantity of light transmitted when the index of liquid and of solid approached each other. We tried this first

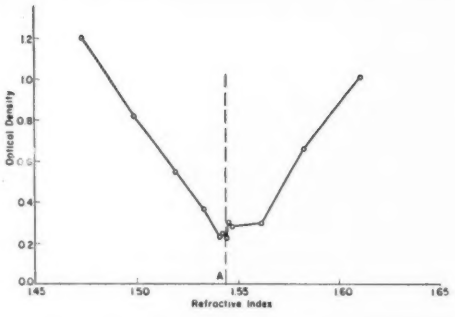

Fig. 6.
Optical density of quartz powder suspensions in liquids of varying refractive index.

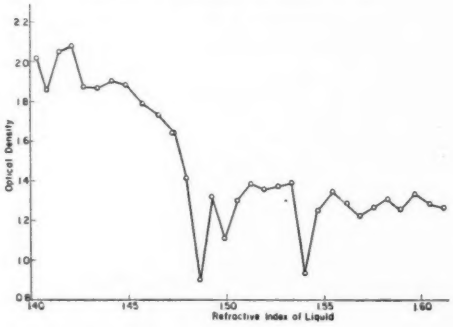

Fig. 7.
Refractive index spectrum for fly ash (No. 1457).

with a powder of known refractive index, i.e., pulverized quartz, and obtained the results illustrated in Fig. 6. The minimum optical density occurred at the index value predicted. Fig. 7 gives the results on a sample of flyash.

It was not possible for us to continue our studies of this technique and it is therefore being mentioned at this time in the hope that others will be interested in developing it. It was found difficult to get reproducible results and some preliminary studies were made involving the time factor (because of sedimentation effects) and solubility in the suspending fluid (mixtures of decalin and alphamethylnaphthalene).

I should like to acknowledge the contributions alluded to in this paper by my present and former colleagues, J. DEANE SENSENBAUGH, GEORGE F. HAINES, JR., HAROLD M. IDE, as well as DR. CHARLES B. WILLING-

HAM and LLOYD J. SULLIVAN who developed the dust classification techniques.

References

1. SENSENBAUGH, J. DEANE, and HEMEON, W. C. L.: Variables in Monthly Dust Fall Measurements. *Am. Soc. Testing Materials Proceedings* 1953, 53:1160. (Reprints available.)

2. HEMEON, W. C. L.: Current Conceptions on Air Pollution. Regional Technical Meetings of the American Iron and Steel Institute, p. 71, 1954. Publication of American Iron and Steel Institute. (Reprints available.)

3. SENSENBAUGH, J. DEANE, and HEMEON, W. C. L.: Unpublished data.

4. HEMEON, W. C. L.; HAINES, JR., GEORGE F., and IDE, HAROLD M.: Determination of Haze and Smoke Concentrations by Filter Paper Samplers. *Air Repair*, 3:22, (August) 1953.

5. HEMEON, W. C. L.; SENSENBAUGH, J. DEANE, and HAINES, JR., GEORGE F.: Design and Use of Equipment in Air Pollution Measurements. *Instruments*, pp. 26, 566, April, 1953.

6. CHOLAK, J.; SCHAFER, L. J.; YEAGER, D. W., and KEHOE, R. A.: Report on the Nature of the Suspended Matter. Report for Air Pollution Foundation, Los Angeles, July 1, 1955.

7. SENSENBAUGH, J. DEANE, and HEMEON, W. C. L.: To be published.

8. HEMEON, W. C. L.; IDE, HAROLD M., and PETERSON, AXEL: To be published.

9. PERKINS, W. A.; LEIGHTON, P. A.; GRINNELL, S. W., and WEBSTER, F. X.: A Fluorescent Atmospheric Tracer Technique for Mesometeorological Research. *Proc. Second National Air Pollution Symposium*, p. 42, 1952.

9a. HOLDEN, F. R.; DRESCH, F. W., and CADLE, R. D.: Statistical Control of Fluorescent Particle Tracer Studies. *AMA Arch. Ind. Hyg. & Occ. Med.*, 9:291-296, April, 1954.

10. WILLINGHAM, C. B., and SULLIVAN, LLOYD J.: Unpublished.

11. SENSENBAUGH, J. DEANE, and HEMEON, W. C. L.: Unpublished.

"Of Publishing Scientific Papers"

BAD writing, according to the familiar aphorism, serves no purpose that cannot be better served by good writing. The thesis applies not only to techniques in writing; it can be expanded to include methods, material and motives. In some two-score pungent paragraphs, employing also the graphic arts in the form of cartoons, DR. GEORGE E. BURCH, Henderson Professor of Medicine at Tulane University School of Medicine, has set up a series of guides for author, reader, critic and editor that indicate right paths by warning against wrong ones. These crystallized concentrates comprise, in their aggregate, a complete short essay under the title "Of Publishing Scientific Papers," published by Grune & Stratton, New York, 1954. Describing a good scientific paper as one that "contains new data well oriented among those already in the literature," DR. BURCH points out that the purpose of the investigation must be indicated, the experiments described and the origin of the ideas precisely presented. The results must then be summarized "clearly but without modification, selection, or elimination of data for the purpose of reaching preconceived, prejudiced or apparently acceptable conclusions." Although the investigator and author is primarily responsible for the quality of the paper, others such as the "benefactor" or sponsor (if any), the institution, the editor and the publisher, all of whom are concerned with its production, must accept their share of the final responsibility.

The essayist heads his blacklist of undesirable characters with the self-plagiarist, a calculating and unfortunately ubiquitous writer who draws freely on his own previous publications for each new paper, continually watering his original porridge into the thinnest of gruels. His bibliography may refer largely to his own published material, and he also quotes from his earlier papers without benefit of quotation marks, but without failing to credit himself with the ideas expressed. Like the self-aggrandizer and the obsessive or quantitative writer, the self-plagiarist, were the recommendations of the Lord High Executioner put into effect, never would be missed.

DR. BURCH has a word or two to say about the responsibilities of the university and other sponsors of research in not prodding for premature publication or using too much the yardstick of publication as the criterion of scientific accomplishment. Likewise the editor and the editorial board, into whose hands is given the selection of papers to be published, should have qualifications that include (so far as possible) "scientific knowledge, irreproachable scientific interest, objectivity, industry, critical judgment and ability to write scientifically themselves." Their interest is genuinely that of good scientific and clinical medicine and the prestige of the journal that they represent. Personal publicity is entirely foreign to the anonymous services that they perform.

—From an editorial in *New England Journal of Medicine*, 252:782 (May 5) 1955.

The Toxicity of the Vapors of Aroclor 1242® and Aroclor 1254®

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INVESTIGATION of the physiological response of animals to the inhalation of two Aroclors, namely 1242 and 1254,® was undertaken because in the earlier literature[1] dealing with the toxic effects of Aroclors, no distinction has been drawn between the toxic effects of chlorinated biphenyls and those of chlorinated naphthalenes, despite the differences in the chemical composition, physical characteristics, and industrial applications of these classes of compounds. In the case of the chlorinated biphenyls, at least, further toxicological investigation and consideration were required to demonstrate the relationship between the extent of their chlorination and their toxicity.

Properties

AROCLOR 1242 is a light, straw-colored, mobile liquid. According to Benignus, of Monsanto Chemical Company, it contains 42.0 ± 0.5% of chlorine, an amount which corresponds to a chlorinated biphenyl with three chlorine atoms in unassigned positions. The physical and chemical properties are given in Monsanto Application Bulletin No. O-P-115. Its specific gravity at 25°/25°C is 1.378 to 1.388; it has a distillation range of 325° to 360°C; a refractive index (D-line at 20°C) of 1.627 to 1.629; a Saybolt Universal viscosity at 100°F of 80 to 93 seconds, and a flash point (Cleveland Open Cup) of 176°-180°C. Its vapor pressure is about 4 mm at 150°C and about 30 mm at 200°C. Although insoluble in water and glycerine, it is soluble in most organic substances. At 745 mm of mercury and 25°C, the concentration of 1 mg of the vapor of Aroclor 1242 per liter of air is equivalent to 96.9 ppm by volume.

Aroclor 1254, which corresponds to pentachlorobiphenyl, is a light, straw-colored, viscous liquid. The positions of the chlorine atoms have not been established. The molecular weight of pentachlorobiphenyl is 326.445, of which 54.3% is chlorine. Benignus has reported that Aroclor 1254 contains 55.0 ± 0.5% of chlorine. Bulletin No. O-P-115 describes Aroclor 1254 as having a specific gravity at 25°/25°C of 1.538 to 1.548; a distillation range of 365° to 390°C, a refractive index (D-line at 20°C) of 1.639 to 1.641, and a Saybolt Universal viscosity at 100° F of 1,800 to 2,500 seconds. The vapor pressure at 150°C is about 1.3 mm, and at 200°C is about 9 mm. The material is soluble in most organic substances, but it is insoluble in water and glycerine. At 745 mm of mercury and 25°C the concentration of 1 mg of Aroclor 1254 per liter is equivalent to 76.5 ppm by volume.

Experimental Method

EXPOSURE TO THE VAPOR: In a preliminary experiment (No. 1) with the vapor of Aroclor 1242, a group of animals was confined for seven hours on each of five days per week in a rectangular plywood chamber (volume 600 liters), of which the inner metal lining was coated with a baked chemically resistant plastic, through which was passed a stream of air laden with Aroclor 1242 in a known concentration. The stream of air, conditioned with respect to temperature (75° ± 3°F inside the chamber), dust and humidity, entered the chamber through an "Anemostat," located at the center of its top, at the rate of 500 liters per minute, as measured by an inclined manometer attached

Presented at the Seventeenth Annual Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, Philadelphia, April 26, 1956.

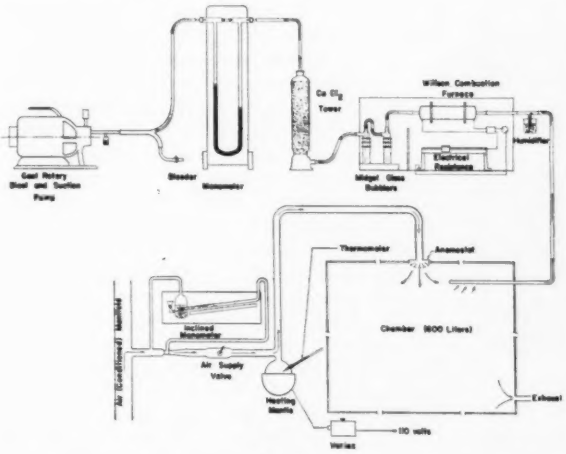

Fig. 1.
Schematic diagram of equipment for volatilizing Aroclor, chamber, and equipment for combustion and collection of samples.

to a venturimeter (Fig. 1). The air was withdrawn by suction from the chamber through an exit tube located on the rear wall near the floor, equidistant from the sides.

Aroclor 1242 was volatilized from a heated glass well maintained at 132° to 138°C. The air passed over the surface of the liquid before entering the chamber (Fig. 1).

In a second similar experiment performed on the same Aroclor (No. 2), the temperature of the liquid was kept at 100° to 105°C.

While the second experiment with Aroclor 1242 was in progress, an experiment involving the vapor of Aroclor 1254 (No. 1) was carried out in a second chamber similar to the first except for the absence of a plastic inner lining. The rate at which air flowed over liquid Aroclor 1254, which was maintained at 130° to 135°C, was 400 liters per minute.

In order to accustom the animals to the experimental procedure, they were kept in their respective chambers for seven hours on each of four consecutive days during the week prior to the introduction of the Aroclor vapor into the chamber. For purposes of control (No. 1), a third group of animals was confined, throughout a like period before and during the period of the exposure to the Aroclors, in another chamber supplied only with conditioned air.

A second set of experiments with each of the Aroclors at lower concentrations was conducted in the same chambers. In the third experiment with Aroclor 1242, which was heated to 55° to 60°C, the air passed over the liquid at the rate of 800 liters per minute. In the second experiment with Aroclor 1254, the liquid was maintained between 115° and 125°C while 700 liters of air per minute passed over its surface before entering the chamber. The animals exposed in each of these experiments, as well as a second group of controls, were confined in separate 600 liter chambers during a seven hour period on each of five days per week during the week preceding the initial exposure of the test animals to the vapor of the respective Aroclors.

In all experiments, the period of exposure or confinement (controls) was seven hours per day on five days per week for several weeks. In all instances, the original group consisted of one cat, six guinea pigs, ten mice, four rabbits, and ten rats. Among both the experimental and control groups, several animals died from extraneous causes during the course of these experiments and were replaced very soon thereafter. In the second experiment with Aroclor 1242, in the first experiment with Aroclor 1254, and in the comparable group of controls, representative animals were killed throughout the experiment.

METHOD FOR THE DETERMINATION OF THE AROCLORS IN AIR: These materials were determined quantitatively by virtue of the fact that, on thermal decomposition, they yield hydrochloric acid which with silver nitrate, forms a suspension, the density of which could be measured by means of the Beckman spectrophotometer at 500 mμ.

On each day, two samples of air from each chamber were collected by passing air at the rate of one liter per minute (for 15 minutes in the case of the higher concentrations and for 45 minutes in case of the lower concentrations) through a fused silica combus-

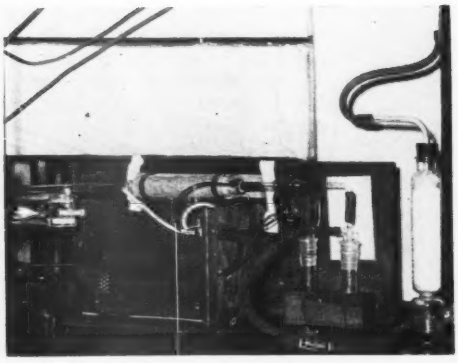

Fig. 2.
Humidifier, furnace, and sampling towers.

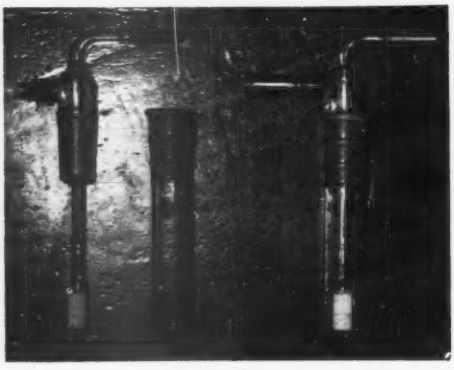

Fig. 3.
Sampling towers. (Midget Bubblers)

tion furnace, and then through two midget bubblers, in series, each containing 10 ml of 0.1 N sodium hydroxide. A quartz tube (13 mm outside diameter, 7 mm inside diameter and 16.25 inches in length) contained several strips of folded platinum foil. The middle nine inches of the tube were wrapped with a heating unit. The heating unit of the furnace (Fig. 2) consisted of 20 feet of B. and S., gauge 22, nichrome wire (1 ohm per foot), and was covered with asbestos cement. The sample of air was humidified by a cotton wick saturated with water placed 0.5 inch upstream from the quartz tube. With the furnace maintained at 850°C, the Aroclor was decomposed in the presence of H_2O to form HCl; this was absorbed in 0.1 N sodium hydroxide in midget bubblers (Fig. 3) equipped with fritted-glass bubblers (Mine Safety Appliance Company No. 43867).

The 0.1 N sodium hydroxide was prepared by dissolving 4.0 g of pellets (ACS specifications 0.01% Cl) in about 50 ml of double-distilled water. To this were added 10 g of arsenic trioxide (chloride-free) dissolved in water and filtered through Whatman No. 42 filter paper. The combined solutions were diluted to 1,000 milliliters.

The sample was transferred to a 25 ml graduated cylinder to which one drop of a solution of phenolphthalein was added (1 g of phenolphthalein dissolved in 100 ml of CP methanol). The solution was neutralized with 3N nitric acid (190 ml CP concentrated nitric acid diluted to 1,000 ml with double-distilled water) and diluted to 23 ml with double-distilled water. One ml of 3N nitric acid was added to obtain a pH of 1. After adding 1 ml of a solution of silver nitrate (3 g of $AgNO_3$ diluted to 1,000 ml with double-distilled water), the suspension was mixed by inversion. After standing 30 minutes, the transmission of wave-length 500 mμ was measured in a 50 cm cell by means of a Beckman spectrophotometer which was set against a corresponding cell containing a reagent blank.

The amount of Aroclor was estimated by means of a standardized curve prepared from known quantities of sodium chloride. A curve presenting the transmission values at 500 mμ of suspensions prepared from sodium chloride ranging from 5.0 to 120 micrograms per 25 ml of final suspension is shown in Fig. 4. On the basis of 42.0% of chlorine in Aroclor 1242 and 55.0% in Aroclor 1254, one microgram of sodium chloride is equivalent to 1.442 micrograms of Aroclor 1242 or to 1.1028 micrograms of Aroclor 1254.

Experimental Results

MORTALITY—AROCLOR 1242: No signs of intoxication were observed in any of the members of a group of 31 animals (Experiment No. 1), all of which survived throughout a period of 24 days, on 17 of which they were subjected to the inhalation of air bearing 8.6 micrograms of Aroclor 1242 per liter (0.83 ppm) for seven hours (Table I).

One cat, four guinea pigs, six mice, two rabbits, and eight rats survived without

signs of intoxication throughout their exposure (Experiment No. 2) for seven hours on each of 82 days over a period of 120 days to air bearing the vapor of Aroclor 1242 in the concentration of 6.83 micrograms per liter (0.66 ppm). Certain other survivors (two guinea pigs, three mice, one rabbit, and two rats) were subjected to fewer periods of exposure (cf. Table I). The deaths from extraneous causes among the exposed animals were fewer than those which occurred among the corresponding group of controls (No. 1, Table I). One cat, three mice, three guinea pigs, three rabbits, and eight rats survived following confinement for seven hours on 84 days over a period of 122 days, in a chamber in which the air, conditioned with respect to dust, humidity and temperature, contained no vapor of either Aroclor. Ten more survivors (three guinea pigs, four mice, one rabbit, and two rats) were subjected to fewer periods of confinement (cf. Table I).

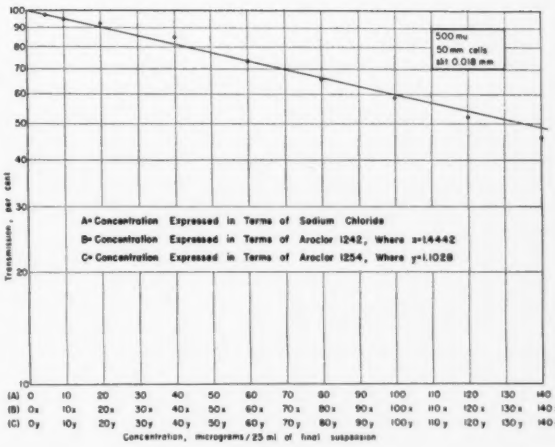
500 mμ
50 mm cells
slit 0.018 mm
Transmission, per cent
A= Concentration Expressed in Terms of Sodium Chloride
B= Concentration Expressed in Terms of Aroclor 1242, Where a=1.4442
C= Concentration Expressed in Terms of Aroclor 1254, Where y=1.1028
(A) 0 10 20 30 40 50 60 70 80 90 100 110 120 130 140
(B) 0x 10x 20x 30x 40x 50x 60x 70x 80x 90x 100x 110x 120x 130x 140x
(C) 0y 10y 20y 30y 40y 50y 60y 70y 80y 90y 100y 110y 120y 130y 140y
Concentration, micrograms/25 ml of final suspension

Fig. 4.
Standard curve for the determination of Aroclor 1242 and Aroclor 1254.

In another experiment (No. 3, Table I-A) in which animals were exposed to air bearing Aroclor 1242 in the concentration of 1.9 micrograms per liter (0.18 ppm) for seven hours per day on 150 days over a period of 214 days, the incidence of mortality among

TABLE I.
SUMMARY OF DATA ON MORTALITY AMONG ANIMALS EXPOSED TO THE VAPOR OF AROCLOR 1242 OR AROCLOR 1254 IN AIR
(Higher concentrations)

Material	Concentration γ/l	Concentration ppm	Duration of Exposure (hours)	Number of Animals that Survived: Cats	Guinea Pigs	Mice	Rabbits	Rats	Expt. No.
Aroclor 1242	8.60	0.83	17 x 7	1	6	10	4	10	1
Aroclor 1242	6.83	0.66	82 x 7	1	$6^{1,1}$	$9^{1,2,2}$	3^{1}	$10^{1,3}$	2
Aroclor 1254	5.40	0.41	83 x 7	1	$6^{1,1,1}$	$10^{1,4}$	$4^{1,3}$	10^{1}	1
Control	0	0	84 x 7	1	$6^{1,1,3}$	$7^{1,5}$	4^{6}	$10^{1,3}$	1

[1]One of the animals was exposed on only 65 to 77 days
[2]One of the animals was exposed on only 41 to 58 days
[3]One of the animals was exposed on only 20 to 33 days
[4]Five of the mice were exposed on only 74 days
[5]Three of the mice were exposed on only 14 to 19 days
[6]One of the rabbits was exposed on only 19 days

TABLE I-A.
SUMMARY OF DATA ON MORTALITY AMONG ANIMALS EXPOSED TO THE VAPOR OF AROCLOR 1242 OR AROCLOR 1254 IN AIR
(Lower concentrations)

Material	Concentration γ/l	Concentration ppm	Duration of Exposure (hours)	Number of Animals that Survived: Cats	Guinea Pigs	Mice	Rabbits	Rats	Expt. No.
Aroclor 1242	1.90	0.18	150 x 7	1	6^{1}	$8^{2,3}$	$3^{1,4,4}$	10^{5}	3
Aroclor 1254	1.50	0.11	150 x 7	1	$7^{2,3,5}$	$10^{2,3,3,6}$	4	11^{3}	2
Control	0	0	150 x 7	1	6^{2}	$10^{1,2,4,5,5,6}$	$5^{1,5,5}$	10^{5}	2

[1]One of the animals was exposed on only 26 to 37 days
[2]One of the animals was exposed on only 41 to 55 days
[3]One of the animals was exposed on only 130 to 139 days
[4]One of the animals was exposed on only 5 to 13 days
[5]One of the animals was exposed on only 107 to 120 days
[6]One mouse was exposed on only 66 days

the various species, with the exception of the rabbits (which died during an epidemic of pneumonia), was no greater than that encountered among a similarly constituted control group (Experiment No. 2, Table I-A).

One cat, five guinea pigs, six mice and nine rats survived throughout the entire period of their subjection to the vapor of Aroclor 1242. Seven additional survivors (one guinea pig, two mice, three rabbits, and one rat) were exposed intermittently to the vapor of Aroclor 1242 over a shorter period of time (cf. Table I-A). Among the group of controls (Experiment No. 2, Table I-A), one cat, five guinea pigs, four mice, two rabbits, and nine rats survived throughout the entire period of confinement, for seven hours on each of 150 days over 213 days in a chamber supplied with conditioned air. Eleven other control animals (one guinea pig, six mice, three rabbits, and one rat) survived during a shorter total period of intermittent confinement (Table I-A). No signs of intoxication were observed among experimental or control animals.

MORTALITY—AROCLOR 1254: One cat, three guinea pigs, four mice, two rabbits, and nine rats survived throughout their exposure, for seven hours on each of 83 days over a period of 121 days, to air containing Aroclor 1254 in the concentration of 5.40 micrograms per liter (0.41 ppm). Certain other animals (three guinea pigs, six mice, two rabbits, and one rat) were killed for examination after 33 to 74 periods of exposure (Table I). The incidence of mortality from extraneous causes among the exposed and control animals (Experiment No. 1), with the exception of the rabbits, was comparable (Table I).

One cat, four guinea pigs, six mice, four rabbits, and 10 rats survived (Experiment 2, Table I-A) following their exposure for seven hours on each of 150 days over a period of 213 days to air containing Aroclor 1254 in the concentration of 1.5 micrograms per liter, (0.11 ppm). Eight other animals (three guinea pigs, four mice, and one rat) survived through 30 to 139 periods of exposure. The incidence of fatalities among the exposed group was slightly less than that encountered among the controls (Experiment 2, Table I-A).

TABLE II.
THE AVERAGE CHANGES IN WEIGHT OF EXPERIMENTAL AND CONTROL ANIMALS
(Higher concentrations)

Species of Animal	Number of Animals	Average Initial Weight (kg)	Average Change in Weight Expressed as Percentage of Initial Weight
Aroclor 1242 - 8.60 γ/l - Experiment No. 1			
Cat	1	2.863	+ 8.1
Guinea Pig	6	0.649	− 2.0
Mouse	10	0.024	+ 9.1
Rabbit	4	3.901	+ 7.7
Rat	10	0.328	+ 2.1
Aroclor 1242 - 6.83 γ/l - Experiment No. 2			
Cat	1	2.271	+22.2
Guinea Pig	6	0.404	+56.4
Mouse	9	0.021	+21.2
Rabbit	3	2.587	+50.5
Rat	10	0.212	+17.6
Aroclor 1254 - 5.40 γ/l - Experiment No. 1			
Cat	1	2.320	+22.1
Guinea Pig	6	0.409	+40.7
Mouse	10	0.021	+28.9
Rabbit	4	2.722	+39.7
Rat	10	0.227	+ 6.9
Conditioned Air - Controls - Experiment No. 1			
Cat	1	2.388	+ 6.6
Guinea Pig	6	0.418	+56.6
Mouse	7	0.022	+17.4
Rabbit	4	2.764	+44.5
Rat	10	0.211	+16.4

TABLE II-A.
THE AVERAGE CHANGES IN WEIGHT OF THE SURVIVORS AMONG THE ORIGINAL GROUPS
(Lower Concentrations)

Species of Animal	Number of Animals	Average Initial Weight (kg)	Average Change in Weight Expressed as Percentage of Initial Weight	P
Aroclor 1242 - 1.9 γ/l - Experiment No. 3				
Cat	1	1.730	+115.0	—
Guinea Pig	5	0.514	+ 43.1	>0.05
Mouse	6	0.0238	+ 21.5	>0.05
Rat	9	0.214	+ 39.9	>0.05
Aroclor 1254 - 1.5 γ/l - Experiment No. 2				
Cat	1	2.858	+ 15.2	—
Guinea Pig	4	0.487	+ 32.5	>0.05
Mouse	6	0.0243	+ 17.0	>0.05
Rabbit	4	2.958	+ 44.4	>0.05
Rat	10	0.222	+ 28.7	>0.05
Conditioned Air - Controls - Experiment No. 2				
Cat	1	3.329	− 7.8	—
Guinea Pig	5	0.500	+ 54.7	—
Mouse	6	0.0257	+ 13.2	—
Rabbit	4[1]	2.622	+ 52.2	—
Rat	9	0.218	+ 33.7	—

[1]Includes two early replacements.

No general or specific signs of intoxication were noted among the experimental animals during or after their exposure.

GROWTH: The pertinent data relating to the changes in weight of the animals of the various groups are presented in Table II and II-A.

Although comparable controls were not weighed during the first experiment with Aroclor 1242 (17 x 7.0 hrs.—8.60 micrograms per liter) all of the animals, except the guinea pigs, appeared to gain normally in weight during the period of exposure. Despite a small net loss by the guinea pigs in this experiment, they were actually gaining weight at the termination of the period of exposure.

The animals exposed to the vapor of Aroclor 1242 in the concentration of 6.83 micrograms per liter (Experiment No. 2) or to Aroclor 1254 in the concentration of 5.40 micrograms per liter (Experiment No. 1), with the exception of the guinea pigs that were exposed to the vapor of Aroclor 1254, grew equally as well as the controls (Experiment No. 1, Table II).

The growth of the surviving experimental animals in the groups initially exposed to the lower concentrations of the vapor of Aroclor 1242 (Experiment No. 3, 1.9 micrograms per liter) and Aroclor 1254 (Experiment No. 2, 1.5 micrograms per liter) was unaffected. No significant differences in the average change in weight of the experimental and the control groups were found by the use of the "t" test (Table II-A).

WEIGHT OF LIVER AND KIDNEYS: The weights of the livers and kidneys and the relationships of their weights to the body weights (expressed as grams per 100 grams of body weight) of the animals that survived following exposure to the vapor of Aroclor 1242 in the concentration of 6.83 micrograms per liter, are shown in Table III, which also gives the corresponding data on the controls. Comparison by the "F" test of the variances of the ratios of the livers or kidneys to the body weights of rats, guinea pigs and rabbits exposed to the vapor of Aroclor 1242 (6.83 micrograms/liter), with those of the controls, revealed no significant differences ($P>0.05$) except in the case of the livers of the rats. Application of the "t" test to the mean values (Table III) revealed no significant differences between the test and control groups of guinea pigs, rats, and rabbits.

Comparable values for rats, guinea pigs and rabbits exposed to Aroclor 1254 in the concentration of 5.40 micrograms per liter

TABLE III.

SUMMARY OF THE DATA ON THE RELATIONSHIP OF THE WEIGHT OF THE LIVER OR THE KIDNEYS TO THE BODY WEIGHT OF ANIMALS EXPOSED TO THE VAPOR OF AROCLOR 1242 OR AROCLOR 1254

Aroclor 1242 - 6.83 γ/l - 82 x 7 hrs.
Aroclor 1254 - 5.40 γ/l - 83 x 7 hrs.
Controls - 0 γ/l - 84 x 7 hrs.

Compound	Organ	Species of Animal	n	Average Organ Weight (g)	Average Body Weight (g)	Ratio of Weight of Organ x 100 to Body Weight	t	P
Aroclor 1242	Liver	Rat	9	11.2	240	4.64	1.2381	0.20-0.30
Aroclor 1242	Liver	Guinea Pig	6	29.6	640	4.68	0.4291	>0.50
Aroclor 1242	Liver	Rabbit	3	122.0	4,023	3.04	1.5085	0.10-0.20
Aroclor 1254	Liver	Rat	10	12.8	239	5.34	3.3992	<0.01
Aroclor 1254	Liver	Guinea Pig	6	25.9	581	4.51	0.0000	>0.50
Aroclor 1254	Liver	Rabbit	4	120.0	3,537	3.53	1.0122	>0.50
Aroclor 1242	Kidneys	Rat	9	1.85	240	0.773	0.5842	>0.50
Aroclor 1242	Kidneys	Guinea Pig	6	5.0	640	0.805	0.7257	0.40-0.50
Aroclor 1242	Kidneys	Rabbit	3	18.6	4,023	0.468	0.4177	>0.50
Aroclor 1254	Kidneys	Rat	10	2.1	239	0.770	0.4199	>0.50
Aroclor 1254	Kidneys	Guinea Pig	6	4.6	581	0.808	0.5411	>0.50
Aroclor 1254	Kidneys	Rabbit	4	15.8	3,537	0.464	0.3163	>0.50
Control	Liver	Rat	10	9.0	243	4.15	—	—
Control	Liver	Guinea Pig	6	30.3	266	4.51	—	—
Control	Liver	Rabbit	4	102.3	3,993	2.61	—	—
Control	Kidneys	Rat	10	2.0	243	0.807	—	—
Control	Kidneys	Guinea Pig	6	4.8	666	0.731	—	—
Control	Kidneys	Rabbit	4	19.5	3,993	0.504	—	—

are also given in Table III. Application of the "t" test to the differences in the mean values which characterized the experimental and control animals (Table III) shows that the weights of the livers of the exposed rats were significantly greater than those of the controls, the ratio of the liver to the body weight of the former being 5.34 g per 100 g of body weight, that of the latter being 4.15 g per 100 grams. In all other instances tested (livers and kidneys of both guinea pigs and rabbits and kidneys of rats) the differences were statistically insignificant.

The organs of the animals exposed to the lesser concentrations of the vapor of the Aroclors were not examined in this manner because of the borderline character of the results associated with the higher concentrations.

LIVER FUNCTION: Data were obtained as to the apparent prothrombin activity of the blood (measured by the method of Kato[2]) of certain animals that had been exposed (1) to the vapor of Aroclor 1242, in the concentration of 6.83 micrograms per liter; (2) to the vapor of Aroclor 1254 in the concentration of 5.40 micrograms per liter; and (3) to conditioned air alone. In Table IV the percentile relationships of the clotting power have been calculated arbitrarily by dividing 100 times the average clotting time of the blood of the control animals by the clotting time of the blood of the experimental animals on the same day. No diminution in the clotting power of the blood, as a measure of the impairment of the function of the liver, was induced by the exposure of the animals to the vapor of Aroclor 1242 in air in the concentration of 6.83 micrograms per liter, or to the vapor of Aroclor 1254 in the concentration of 5.40 micrograms per liter.

This functional test was not applied to animals subjected to the lower concentrations, because of the negative results obtained when the animals were subjected to the higher concentrations.

HEMATOLOGICAL RESULTS: Determina-

TABLE IV.

THE EFFECT OF EXPOSURE TO THE VAPOR OF AROCLOR 1242 OR AROCLOR 1254 IN AIR FOR SEVEN HOURS PER DAY ON FIVE DAYS PER WEEK OVER A PERIOD OF SEVERAL MONTHS UPON THE APPARENT PROTHROMBIN CONTENT OF THE BLOOD OF CATS AND RABBITS

Relation of "Prothrombin Time" of Control and Exposed Animals Expressed Arbitrarily in Terms of Percentage Change in Clotting Time of Blood 100 x Clotting Time of Blood of Control Animals Clotting Time of Blood of Exposed Animals		Time of Sampling in Relation to Period of Exposure	
		Days Before First Period	Serial Number of Period After Which Blood Was Drawn
Aroclor 1242 - 6.83 γ/l - Experiment No. 2			
Cat A-445	Rabbits (Average)		
100.0	89.6	3	
90.3	93.8	2	
113.6	105.1		10
101.5	99.0		30
97.4	98.0		45
116.2	103.7		59
	101.5		82
	100.0		[1]
Aroclor 1254 - 5.40 γ/l - Experiment No. 1			
Cat A-444	Rabbits (Average)		
100.0	97.1	3	
100.0	96.9	2	
	81.8		9
98.5	90.0		10
	99.6		15
113.6	102.0		30
	112.1		32
101.5	97.0		45
92.5	95.4		59
90.0	95.2		83
	100.0		[2]

[1]Determination made 15 days after 82nd period of exposure.
[2]Determination made 14 days after 83rd period of exposure.

TABLE V.
THE AVERAGE NUMBERS OF ERYTHROCYTES AND LEUCOCYTES AND THE AVERAGE CONCENTRATION OF HEMOGLOBIN IN THE PERIPHERAL BLOOD OF EXPERIMENTAL AND CONTROL ANIMALS

Compound	Concentration (γ/l)	Experiment Number	Species of Animal	Erythrocytes: (Thousands) per mm³	Leucocytes: per mm³	Hemoglobin: g/100 ml
Aroclor 1242	6.83	2	Guinea Pig	5,701	10,112[1]	14.4[2]
			Rabbit	6,088	9,755	12.1
Aroclor 1254	5.40	1	Guinea Pig	5,886	12,927	14.5[2]
			Rabbit	6,486[2]	10,811	13.2
Controls	0	1	Guinea Pig	5,729	12,124	13.9
			Rabbit	5,909	11,590	12.2

[1]Value significantly less than that yielded by controls.
[2]Value significantly greater than that yielded by controls.

tions of the numbers of erythrocytes and leucocytes and of the hemoglobin content in the peripheral blood of the guinea pigs and rabbits subjected to inhalation of the vapor of Aroclor 1242 in the concentration of 6.83 micrograms per liter, are given in Table V, which also includes comparable results on control animals. Application of the "t" test to differences in the mean values for the experimental and control animals yielded borderline evidence of significant differences in the number of leucocytes and in the concentration of the hemoglobin in the blood of the guinea pigs. These differences were small and of opposite sign (the numbers of leucocytes in the blood of the test animals were low, while the hemoglobin content was high), as well as being subject to individual variations, and they cannot be regarded as of physiological significance. Somewhat comparable results were obtained when guinea pigs and rabbits were subjected to Aroclor 1254 in the concentration of 5.40 micrograms per liter. No physiological significance is attached to the slightly elevated hemoglobin content of the guinea pigs.

PATHOLOGICAL FINDINGS: In all of the experiments, animals were killed from one to 15 days after the final period of exposure.

In the first experiment with Aroclor 1242 (8.60 micrograms per liter), all of the animals were examined postmortem, and since gross examination of the viscera did not reveal any significant alterations, the tissues of only representative animals (one cat, four guinea pigs, six mice, four rabbits and six rats) were sectioned and examined microscopically. No abnormalities were found in the viscera of these animals.

In the second experiment with Aroclor 1242 (6.83 micrograms per liter), and in the first experiment with Aroclor 1254 (5.40 micrograms per liter), which were carried out simultaneously, the deaths that occurred among the test and control animals were the apparent result of an appreciable incidence of pneumonia. The lesions of certain of the animals were those of frank pneumonia; in others such lesions were not fully developed. Degenerative changes in the viscera were usually found, in varying degrees of severity, in association with the pneumonia, but in certain animals, test and control alike, the degenerative changes were more evident than the pneumonia. The similarity of the lesions in test and control animals, and the lack of characteristic evidence of chemical pneumonitis, led to the reasonable, but not altogether certain, conclusion that all of these fatalities resulted from intercurrent disease among the animals, and not from the effects of their exposure to the Aroclors. For practical purposes, this conclusion was subjected to the critique of further experiments involving more prolonged exposure of animals to somewhat lower concentrations. Except in the case of the rats exposed to Aroclor 1254, the survivors subjected to the higher concentration of either Aroclor had normal viscera.

All of the animals exposed to the vapor of Aroclor 1242 in the concentration of 1.9 micrograms per liter were examined postmortem, and the viscera of most of them were examined microscopically. The few deaths among the exposed group were attributed to incidental infectious pulmonary disease. All exposed survivors, except two rabbits that had hepatic lesions of coccidiosis, had normal viscera. The control animals (Experiment No. 2) that died were found to have had pneumonia. Of those that sur-

vived, one rat, two guinea pigs, and one mouse had focal or diffuse cytoplasmic vacuolation of the hepatic cells. The remaining control animals had normal viscera.

All of the animals exposed to the vapor of Aroclor 1254 in the concentration of 1.5 micrograms per liter were examined postmortem, and the viscera of most of them were examined microscopically. A guinea pig that died exhibited chronic pyelonephritis, pulmonary hyperemia and edema, and degenerative lesions in the brain and liver. The visceral lesions were related, no doubt, to the renal infection. The deaths of four mice were attributable to acute bronchitis and pneumonia. The viscera of the cat that survived were normal. Of the seven guinea pigs that were living when the experiment was terminated, three had normal viscera and four had slight alterations of hepatic cells characterized by cytoplasmic vacuolation. Ten mice survived, and of these six had normal viscera and four had slight degenerative changes in the liver. Four rabbits killed one to 15 days after the last period of exposure had diffuse hepatic degeneration. The character of the lesions varied from cloudy to hyaline or hydropic degeneration and included varying degrees of fatty metamorphosis. The other viscera of these animals were normal. All of the rats were examined and found to have slightly to moderately severe degenerative lesions of the liver. The lesions of greatest severity were found in the rat that was killed and examined on the first day after the last period of exposure. Two rats had chronic pyelonephritis, and the remainder had slight degeneration of the renal tubules.

Discussion: In terms of mortality, growth (except that of guinea pigs) and non-occurrence of pathological changes, the vapor of Aroclor 1242 in the concentration of 8.6 micrograms per liter (approaching saturation) appeared to be non-injurious to experimental animals subjected thereto for seven hours on each of 17 days over a period of 24 days.

Experimental animals subjected to but a slightly lower concentration of Aroclor 1242 (6.83 micrograms per liter) for seven hours per day on each of 82 days over the period of 120 days suffered no injury on the basis of any of the following criteria: mortality, growth, pathology, organ enlargement, liver function or hematological changes.

More prolonged exposure of animals to a lower concentration of Aroclor 1242, (1.9 micrograms per liter) over the period of seven months was likewise without harm in terms of growth, mortality and the absence of pathological changes.

In view of this evidence it is suggested that the tentative allowable concentration of the vapor of Aroclor 1242 should be at least two micrograms per liter (2 mg per cu meter), which is twice that recommended by the American Conference of Governmental Industrial Hygienists[3] for a chlorinated diphenyl of unstated chlorine content.

The exposure of animals to the vapor of Aroclor 1254 in the concentration of either 5.40 or 1.5 micrograms per liter failed to induce harmful effects in the form of retardation of growth (except in the case of guinea pigs exposed to the higher concentration), or of mortality, but histopathologic evidence of apparently reversible hepatic cellular injury was found in the animals. These findings cannot certainly be attributed to the effects of Aroclor 1254 because of the appreciable incidence of pneumonia among both experimental and control animals. When these nonspecific toxic changes in the viscera of the animals were associated with pneumonia, they were readily explained thereby, but they were also found in animals that had been exposed to the vapor of Aroclor 1254 and were free of pneumonia. That these may have represented toxic effects of exposure to Aroclor 1254 finds support in the fact that the livers of the exposed rats (5.40 micrograms per liter) were significantly heavier in relation to their body weight, than were those of control rats. It would appear that this material, which is reported to contain 55% of chlorine, is somewhat more toxic than is Aroclor 1242, which contains only 42% of chlorine. Therefore it is suggested that the threshold concentration of 1 mg per cu meter of air recommended tentatively for safe industrial practice by the American Conference of Governmental Industrial Hygienists[3] is reasonable.

It should be noted that it was necessary to heat these Aroclors in order to increase the rate of volatilization sufficiently to attain the concentrations maintained in these

experiments. To the extent that their industrial usage is carried out at ordinary temperatures, the hazard of their inhalation may well be slight or entirely absent.

Summary

PROLONGED intermittent exposure of animals to the vapor of Aroclor 1242 (1.90 to 8.63 micrograms per liter) demonstrated no injury. Prolonged exposure to comparable concentrations of Aroclor 1254 resulted in reversible degenerative changes in certain viscera.

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References

1. DRINKER, C. K.: Further Observations on the Possible Systemic Toxicity of Certain of the Chlorinated Hydrocarbons with Suggestions for Permissible Concentrations in the Air of Workrooms. *J. Ind. Hyg. & Tox.*, 21:155, 1939.
2. KATO, K.: Micro-prothrombin Test with Capillary Whole Blood; Modification of Quick's Quantitative Method. *Amer. J. Clin. Path.*, 10:147, 1940.
3. American Conference of Governmental Industrial Hygienists. Threshold Limits for 1955. *AMA Arch. Ind. Health*, 11:521, 1955.

American Sanitary Engineering Intersociety Board

AFTER almost ten years of hard work on the part of a handful of interested and determined persons, the American Sanitary Engineering Inter-society Board has become a reality. The ASEIB is an incorporated organization sponsored by the American Society of Civil Engineers, the American Society for Engineering Education, the American Public Health Association, the American Water Works Association and the Federation of Sewage and Industrial Wastes Associations. The purpose of the Board is to improve the practice, elevate the standards, and advance the cause of sanitary engineering. The term "sanitary engineering" is employed in the broad sense as defined by the National Research Council, and embraces the entire concept of engineering biotechnology application for control of environmental stresses. As most readers know, this definition includes industrial hygiene, air pollution control and radiological health. The Board of Trustees of the ASEIB is made up of three representatives from each of the sponsoring bodies and three elected at large by the Board. We are happy to announce that two of the Trustees at large are industrial hygiene engineers, namely LT. COL. ALVIN F. MEYER, JR., Headquarters Strategic Air Command. and ALLEN D. BRANDT, Bethlehem Steel Company.

One of the most important functions of the ASEIB will be examining qualified licensed professional engineers and granting certificates of special knowledge in sanitary engineering or one of its specialties to those who show acceptable competency in the examination. In this respect the Board will function in a manner not unlike that of the American Boards of medicine. For an initial period, outstanding members of the profession having not less than 15 years acceptable experience will be granted certificates without examination. Persons who have been certified by the board will automatically become members of the American Academy of Sanitary Engineers which is the register of holders of such certificates.

The board was incorporated in October, 1955. At the present time the Chairman of the Board is PROFESSOR EARNEST BOYCE, University of Michigan; Vice-Chairman is W. A. HARDENBERG; Treasurer is R. S. RANKIN; Secretary is FRANCIS B. ELDER. It is contemplated that applications for certification will become available in June. All qualified industrial hygiene engineers, air pollution control engineers and radiation safety engineers having not less than eight years experience in this work are urged to apply for certification. The benefits that will result from this movement and the advantages that will accrue to all who become certified are many. What is more, they will increase with the passing years. Application forms may be obtained from the Secretary, FRANCIS B. ELDER, 33 West 39th Street, New York 18, New York, or from members of the Board of Trustees.

Radiation Protection

INSURANCE AND INDUSTRIAL RELATIONS ASPECTS

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CHAPTER, 6, Section 53, of the Atomic Energy Act of 1954 states, "(8) The licensee will hold the United States and the Commission harmless from any damage resulting from the use or possession of special nuclear materials by the licensee." This simple sentence has created one of the most difficult hurdles in the way of the development of nuclear energy for peacetime usage. It has caused industry to examine its potential liability from operation of nuclear reactors and to attempt to evaluate the kinds of insurance protection which would be needed. Fantastic stories have been developed about all of the terrible things which could happen if a nuclear reactor should fail. If losses of the magnitude described could occur, they would represent a national catastrophe. Thus the insurance phase of the reactor program has attracted national interest.

Kinds of Insurance

THERE ARE many kinds of insurance involved. In the casualty field, Workmen's Compensation provides indemnification to employees for time lost and medical costs resulting from industrial injury and occupational disease. In all states, employers must provide such financial protection to their employees. Limits of liability are established by law and thus it is possible for an insurance carrier to estimate its potential maximum liability. The charges for such coverage are based on payroll and are determined on the basis of experience of each kind of operation. Rates in many cases are modified in individual plants by experience. Thus an incentive is provided for the reduction of accidents and occupational disease.

A second kind of insurance of interest in the radiation field is that of providing financial protection against damage to the physical plant. This would include loss by fire, breakdown of equipment, boiler damage, and the like. There has been much discussion about whether or not radiation contamination will be covered under fire policies. As in the case of Workmen's Compensation insurance, the potential loss can be estimated, since the value of all equipment and buildings is known. In fire insurance, for example, the limit of the carrier's liability is stated in the policy.

The serious stumbling block in the dilemma of insurance for reactors is in the field of public liability. This kind of insurance provides financial protection against loss caused to the public, in any way, as a result of plant operations. It is obvious that it is virtually impossible to predict the potential maximum loss which can occur. The severity of such a loss is determined by the number of people who could be injured and the amount of property which could be damaged. Who would care to estimate the maximum amount of damage which could be caused by a serious reactor failure in a densely populated area? As in the case of fire insurance, the carrier limits its liability by the value of the policy. The insurance problem in the reactor field is caused by a wide discrepancy between the amounts of liability insurance which industry feels it needs and the maximum limits which insurance carriers can provide.

In addition to the major insurance coverages discussed above, there are a few other problems. One of these is business interruption coverage for situations where a reactor failure might result in a power plant

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shutdown with a resultant loss of production by plants forced to cease operations because of lack of power.

Another serious problem is the possibility of compounding losses. In a case where a defective component in a reactor failed, causing damage to the reactor with subsequent public liability losses, the owner of the reactor would attempt to recover his loss from the equipment manufacturer. In cases of this type, costly litigation generally results. This could mean that every manufacturer of every component of reactors should have very large public liability limits. Because of this, attempts are being made to find methods for financial protection for reactor operations on a package basis, so that the complications described can be avoided.

Historical

SINCE about 1912, industry and the medical profession have been applying methods utilizing radiation. This 40-year period can be broken down into three periods. The first of these may be referred to as the pre-Manhattan Project era in which the kinds of exposures involved such things as industrial radiography, the medical uses of both radioactive materials and x-ray; the use of self-luminous paints (with very bad experience) and the manufacture of gas mantles using thorium compounds. Problems of coverage were relatively simple because these exposures were generally a normal phase of each specific industry and were, therefore, part of the normal experience. There was some adverse experience but by and large these uses of radiation posed no particular insurance problems.

In brief, it can be stated that during this pre-Manhattan Project era problems of indemnification for loss from radiation injury were minimal and handled in a rather routine fashion.

The second period we may call the Manhattan Project era. This might be referred to from the standpoint of those on the outside as the "great hiatus." During this time security reigned and there were no insurance problems, since the Federal government assumed responsibility for all liability for injury or damage resulting from any operations within the scope of the program. Insurance policies were written by the carriers and the mechanics of handling and settling claims were provided by the carriers, but they were reimbursed in full for all losses.

The next step, historically, was to move into the post-Manhattan Project era which saw the beginnings of the Atomic Energy Commission. During this period which lasted until the passage of the Atomic Energy Act of 1954, security was still the byword, but the light began to dawn. With the advent of the Atomic Energy Commission the insurance picture within AEC installations remained unchanged. New problems began to appear, however. For example, a wide variety of isotopes became available for research as well as for medical and industrial applications. One of these new problems included that of developing equitable insurance rates for these new exposures in the light of a lack of experience. Another was that of delay in onset of radiation damage where the question was raised as to the kind of liability being accumulated today to be paid for 20 years from now. Another question frequently discussed was the adequacy of the benefits under present laws for higher salaried technical personnel. Finally the question was raised as to possible new kinds of radiation and unknown effects which might develop. By and large, all of these problems stemmed from two things. One was the very limited public information on matters relating to exposures to some of these radioactive materials. The other was the fact that many more people were being exposed in a much broader group of industries.

During this time there was a considerable amount of confusion, particularly in the insurance industry as to how to handle this new problem. Attempts were made during 1949, with the establishment of a Joint Casualty Committee on Radiation, to find an answer. The purpose of this committee was to examine the entire problem, particularly as related to the increased use of radioactive materials and to try to set up some way of rating these new exposures. A tentative procedure was drawn up, based on the major premise that radiation damage is an occupational disease. A rather involved proposal was considered in which external radiation sources (both fixed and portable) and radioactive materials were subjected to a

complex rating and credit system. Since the industrial applications of radioactive materials were relatively slow during this time experience was accumulated in the field and fortunately the rating schedule never saw the light of day. At the present time, industrial and medical uses of radioisotopes carry no special rates. Each industry establishes its own experience, good or bad, which will ultimately be reflected in the insurance rates for the industry.

Reactor Problems

THIS BRINGS us to the present. It brings us to the entry of industry into the nuclear reactor field. It brings us to the Atomic Energy Act of 1954 which placed squarely upon the shoulders of American industry the financial responsibility for any damage or injury which might result from the operation of nuclear reactors.

Nuclear reactors, whether for research, medical or power application provide some interesting insurance questions. The developments have come so fast that it is almost impossible to keep abreast of them. Industry has felt that since under certain circumstances reactor failure could cause very severe losses, particularly in areas surrounding the reactor, they were taking on potential liabilities which in some cases could exceed their resources. They felt the need of financial protection. Their prime concern was in the field of public liability. It was felt that if large amounts of fission products were dumped on a community as a result of a run-away reactor the losses could run into hundreds of millions or even billions of dollars. Because of this, industry began a search of the possibilities of obtaining the kinds of protection which they felt were needed from insurance carriers.

They found that the insurance industry was no more anxious to risk its very existence on these experimental operations than was industry. In the first place, there are legal limits on the amount of insurance coverage which can be provided a single policyholder. This is not a problem in the field of Workmen's Compensation insurance. The really serious questions arise from the possible large and unpredictable losses under the heading of public liability. It has been suggested that a serious accident could involve injury and death to people living and working in the area around the reactor operations as well as contamination by radioactive materials of structures in the vicinity. Industry appears to feel that such losses could be extremely large.

As has been pointed out, the carrier in providing public liability insurance restricts its liability by the financial limit set forth in the policy. In such an event the insurance carrier is responsible for losses up to this figure and the insured is responsible for losses above this figure. In other words, the policy specifies the limits of liability which the insurer is willing to accept. In the case of reactor operations the limits which the insurance carrier can accept are related to many factors. They are at the present time considerably less than those which industry appears to want.

During normal operations of a reactor there are no problems which seriously bother informed insurance underwriters. There appears to be no problem of any consequence related to Workmen's Compensation coverage.

A relatively large number of reactors have been operated for several years. By and large, they have compiled excellent safety records. Since all of these were Government installations there was no compromise with safety. Very large sums of money were spent in order to assure that there be no injury to operators or the public. Questions have been raised as to whether or not this same philosophy will continue to be in force, particularly in the field of the operation of power reactors when competition enters the field.

The insurance industry has been studying this problem energetically for some time. A committee of insurance executives has been provided with the necessary security clearances and has been thoroughly indoctrinated by the Atomic Energy Commission so that they could understand all phases of the problem. Since their education has been completed, the insurance carriers have been devoting a considerable amount of time and effort to develop a program which will provide the maximum amount of coverage possible by the world insurance market. The numbers which have been proposed for limits of liability are still far short of those which industry seems to want. This has raised the question as to just how additional

protection will be provided. One of the most popular approaches is to have the Government provide some sort of protection above the limits which the insurance carriers will be capable of supplying. This whole matter is actively under debate, both within the Atomic Energy Commission and by the Congress. What the outcome will be it is impossible to predict.

Protection Problems

LET US EXAMINE the problems of protection which face not only the insurance industry, but all of us who are involved in the field of industrial health.

There are a great many factors that have to be considered in estimating the possibility of loss and the potential cost of such in reactor operations. Of course, each individual reactor must be evaluated separately. The several criteria which must be applied are: (1) the use to which the reactor is to be put; (2) reactor type and design; (3) location of the reactor; (4) amount of containment; (5) quality of the supervision and operating personnel; and (6) the safety program and inspection system.

Reactor Use

AT THE PRESENT time reactors are being considered for research, testing of materials, production of power and propulsion. The degree of risk will depend partly upon which of these uses is contemplated. Most presently proposed *research* reactors are to be built by universities, hospitals or other research organizations. They may be used for undertaking nuclear research, for training personnel in reactor design and operation, or for medical research and treatment. Reactors in this category will probably be relatively small and because of their use will be designed for extreme flexibility. Large numbers of persons, many of them inexperienced, will be operating these installations.

Since every known reactor accident to date has been caused by human error, it is obvious, that for this kind of reactor where many persons may be involved in its operation, the safety will depend more on the quality of the supervisory personnel than it will on the design of the reactor. From the standpoint of the public exposure, losses could be minimized by remote location. Reactors of this type cannot, however, be used effectively unless located in centers of population. A reactor for a hospital is of little use many miles away. If the reactor is to be used for teaching purposes it must be located convenient to the students.

Because of their small size, reactors of this type will probably have a limited hazard as far as damage to the public and contamination are concerned. But because of the nature of the operation and the large number of persons involved, this type of reactor will require not only careful evaluation, but constant supervision.

The focus of attention is the development of nuclear reactors for *power* purposes. The objective of this whole program is to encourage private industry to enter the nuclear power field in the hope that it will be accelerated. There are several companies and groups of companies actively working in this field. There are many different kinds of reactors under consideration. At this stage of the development of the program it can certainly be considered as experimental. Because of their size and the amounts of radioactive material which may be present, the potential, particularly in the way of radioactive contamination, is large. The possibility of a reactor failure, however, is probably not so great in most of the power reactor types as is the case with research reactors. The reason for this is that the primary purpose in building these reactors is for the production of power. Therefore, it is expected that they will be operated more or less continuously at reasonably uniform power levels and with a stable staff. Thus the possibility of an incident is lessened but the potential damage increased for reactors of this type.

Little is known at the present time outside the Atomic Energy Commission about *materials testing* reactors. Their important function will be to assist industry in testing the effects of radiation on various kinds of materials to be used in the construction of power reactors. They should have a stable staff and be classified probably between research and power reactors as far as flexibility is concerned.

Reactors for *propulsion* purposes are still being developed solely under the control of the Atomic Energy Commission and so insurance problems do not exist at this time.

Reactor Types

ONE of the objectives of the current industrial reactor program is to attempt to find the kinds of reactors which will produce power most economically and with a minimum of hazard. Therefore, during this development stage there will be many different kinds of reactors designed and built. Each of them will have its own peculiarities and the degree of hazard will vary widely from type to type. Safety in design will be a criterion of utmost importance in the evaluation of any reactor.

Reactor Location

AS HAS BEEN indicated above, the degree of the hazard or the loss potential is related directly to the number of people and the amount of property which can be damaged. This is an important aspect in evaluating the size of losses which can occur from a reactor incident. The obvious way out is to suggest that all nuclear reactors be located in isolated places. This was exactly the approach taken by the Atomic Energy Commission in setting up its experimental reactor testing facilities in Idaho. If reactor installations are to be of any public use, however, they cannot be located in areas completely isolated from people. In determining liability limits insurance carriers must examine in great detail the neighborhoods or communities in which reactors are to be located so that they can estimate the loss potential.

Containment

SINCE it is not feasible to isolate these installations it has been suggested that neighborhood contamination by radioactive materials can be reduced substantially or eliminated by complete containment, either of the reactor core or of a portion of the building housing the reactor. While this may provide a suitable substitute for isolation of the reactor it does not appeal particularly to those proposing to build such installations because of the increased financial burden in providing for suitable containment. Nevertheless, it seems reasonably certain that if we can in fact have contamination of the magnitude that has been suggested by industry they must accept either isolation or containment as a means of limiting the exposure if they expect maximum participation by the insurance industry in a program of financial protection.

Personnel

SO FAR the discussion has been confined to the physical aspects of the reactor itself. Even more important, however, is the quality of people who will be operating any reactor. Practically all reactor incidents reported to date have been the direct result of some human failure. Carelessness or ignorance can offset the most careful design and safety devices.

It is a well established principle in the field of industrial safety and industrial hygiene that the effectiveness of health and safety programs are in direct proportion to the interest of the management. Thus, in addition to well-trained and conscientious operating personnel a management which establishes an adequate health and safety organization and gives its full support and backing to this organization is an absolute "must."

In addition to the problems of operating the reactors themselves we are beginning to face some of the problems related to ancillary operations. Today we are concerned with plants engaged in the construction of fuel elements for reactors yet to be built. The problems associated with removal and shipment of spent fuel for reprocessing will have to be met. There are problems in public liability associated with an accident to a vehicle transporting spent fuel; there are problems in storage of such fuels prior to shipment. As the program gets underway it is inevitable that industry enter the field of fuel processing and private industry will have its Public Liability problems magnified.

Waste Disposal

THE AMOUNTS of radioactive wastes will vary with the size and type of reactor. In some types there will be problems related to both air and stream pollution. These will both influence discussions on public liability insurance coverage and rates.

Disaster Planning

IN SPITE of what may appear to be adequate control measures, a reactor failure is always a possibility. A complete well-conceived disaster plan, including evacuation,

treatment of injured and decontamination must be set up. Failure to do this can lead to chaos, panic and increased damage in the event of an accident.

Other Radiation Problems

WHILE our attention has been diverted by the more spectacular phases related to nuclear reactors, there has been a fairly rapid expansion of the use of radioactive materials and high energy particle accelerators in both industrial and medical fields. The two important consequences of this growth in terms of insurance problems are, first, the ever-increasing number of people coming in contact with radioactive materials and, second, the substantial increase in both the amounts of radioactivity and the energies involved.

As has already been indicated we have had many years of experience in the medical field and the technique for approaching and solving these problems is reasonably well established. However, the internal use of isotopes for diagnosis and treatment of some diseases poses public liability and malpractice problems but to date the experience in these areas has been good. By and large, from an insurance standpoint this can be handled in a normal and routine fashion.

In industry, one of the significant developments has been the introduction of multicurie sources of isotopes for radiographic purposes. Enough information is available to permit this work to be carried on safely if the rules and regulations are followed.

The use of such things as thickness gauges and other techniques involving radioactivity which are used for process control pose problems in insurance related to public liability. The principal thing with which we are concerned in this regard is the possibility of a failure of the control mechanism resulting in spoilage of materials. There are at the present time hundreds of such devices as beta ray thickness gauges in use, with no serious problems.

Waste disposal problems in the industrial use of radioisotopes has been minimal at the present time.

Industrial and Public Relations Problems

BECAUSE of the highly technical nature of this problem and the fact that atomic energy is generally associated in the public's mind with nuclear weapons, industry's relations with both its employees and the public are extremely critical. Industry must learn to deal with this problem in the open. Attempts at concealing the facts of the use of radiation may only lead to suspicion and distrust. Many companies have already learned that failure to inform the public in detail prior to embarking on operations of this type frequently leads to spirited and sometimes successful opposition. Those plants which have openly discussed their plans with local public officials and with the public through intelligent newspaper publicity have had little or no difficulty. In dealing with one's employees it is equally important that they be appraised of all potential hazards, told of the safeguards which are being taken and trained to do their job safely and effectively. Failure to follow these three simple steps can also lead to suspicion and misunderstanding.

Recently a group of labor leaders presented to the Congressional Joint Committee on Atomic Energy a series of proposals to safeguard workers in the atomic energy field. In the area of health and safety their recommendations included provisions for adequate protective equipment, proper shielding, good monitoring and training of employees in health and safety. They also called for equitable compensation for employees who might be injured by radiation. All of these are certainly justified requests. If industrial management provides all necessary safeguards and the employees carry out their responsibilities in living up to safety rules and regulations there should be no serious problem.

Relation to Radiation Protection

WITH this perhaps over-simplified statement of some of the problems related to our expanded activities in the field of nuclear energy it is easy to see that insurance carriers must have more than a financial interest in this entire problem. No one will deny that it is to everyone's interest to make certain that these operations are carried out safely. These developments will provide an opportunity of almost unlimited scope for specialists in the field of industrial hygiene. Industrial hygienists in industry, governmental agencies and insurance car-

riers are going to have a job ahead of them of tremendous magnitude.

During the first few years of this program as industry and others gain experience in reactor operations the people trained in the field of industrial health are going to be called upon to play their part in preventing radiation and other types of injury. It has been recommended that the Atomic Energy Commission provide an inspection system to follow these plants as they develop. The activities of such groups as the Advisory Committee on Reactor Safety should certainly continue an active interest in all plans and specifications as well as location of all reactors. There will also be industrial health problems in fuel fabricating plants which must be supervised.

Never before has so great a challenge been made to industrial hygienists. While it is an excellent idea to have full financial protection, it is much more important to prevent any such incidents as those which have been discussed.

The viewpoint of industrial hygienists will be essential during the design as well as during operation of reactors. Detailed programs for air sampling, monitoring, personal protection, ventilation design and maintenance, control of atmospheric and liquid wastes, handling and storage of fuel, handling, storage and transportation of spent fuel and disaster planning will all require the broad outlook and experience of industrial hygienists. As this work progresses, the problems will spread. They will appear in many new kinds of plants. It is difficult to predict where one will find exposures related to the reactor program or to other uses of radioactive materials.

Another important contribution which can be made by industrial hygienists is in the fields of public and industrial relations. First we must do a thorough job of providing essential safeguards and then we can provide the facts and assurances which must be given to the public and to employees. We can also contribute to the training and education of employees so that they can play their part in securing safe operation.

In summary, the problems created by the spreading activities in nuclear energy and related fields cover many areas. Activities of industrial hygienists can contribute substantially to solution of these problems in insurance, public relations and industrial relations. Experience is going to determine the future of many of these programs involving radiation. If we do a thorough job so that the experience is good, the private development and use of nuclear energy for peaceful uses will make rapid progress and industrial hygiene will have made a major contribution.

Research Grant

THE PUBLIC HEALTH SERVICE has announced a new procedure to expedite the processing of research grant applications for those requests which do not exceed $2000.00 plus indirect costs and which do not ask support for more than one year. Such applications will be accepted and processed on receipt and are not therefore subject to the usual deadlines for submission prior to review. Council recommendations can be expected on these applications within one to four months from the time of submission. These procedures do not apply for requests for supplements to existing grants. All applications as well as requests for forms or additional information should be addressed to the Division of Research Grants, National Institutes of Health, Bethesda 14, Maryland.

Determination of Acetic Acid in Air

FRANKLIN MILLER, RICHARD SCHERBERGER,
HENRY BROCKMYRE, and DAVID W. FASSETT, M.D.
Eastman Kodak Company, Rochester, New York

ONCE KNOWN only as a constituent of vinegar, acetic acid now plays an important role in the synthetic chemicals industry. Large quantities are used in the production of various cellulose acetates, the plastics upon which some photographic emulsions are coated. It is used in the manufacture of organic dyes, white lead, pharmaceutical chemicals and as a solvent for many organic reactions. By the year 1953, the annual production in the United States of both natural and synthetic acetic acid had risen to 244,000 tons and even in the mild recession of 1954, this dropped to only 225,000 tons.[1]

The increasingly wide and varied use of acetic acid has highlighted a need for better methods of determining air concentrations of this chemical. In addition, our knowledge of acetic acid must be increased by more specific environmental and medical data.

Originally, the method described in this paper was developed to determine air concentrations of acetic acid in organic chemical laboratories and in photographic processing rooms. A variation of this technique has also been used to determine air concentrations of ammonia and some organic amines.

Toxicity of Acetic Acid

AQUEOUS SOLUTIONS of acetic acid can produce severe skin and eye burns; this is especially true when solutions stronger than 80% are used. Acetic acid vapor has a characteristically pungent odor and in higher concentrations causes lachrymation. Inhalation of the fumes can result in temporary irritation of the mucous membranes of the nose and throat.

The authors found air concentrations of 50 ppm to be tolerable for only a few minutes, since this concentration produced tears and intense temporary irritation of the nose and throat. These symptoms disappeared shortly after leaving the contaminated area. The minimum irritating concentration was not studied, although some acclimated workers appear to tolerate up to 30 ppm with no symptoms of irritation. The recommended maximum acceptable concentration is 10 ppm.[2]

Problems of Measurement

NO SPECIFIC methods for determining air concentrations of acetic acid vapor were found in the technical journals. Feigl[3] mentions several micro tests for determining acetic acid qualitatively. None appeared applicable to air sampling. The most widely used method for determining acid air contaminants appears to be absorption in dilute caustic solution and subsequent titration with dilute acid. This method is accurate and shows high absorption efficiency, but the sample must be returned to the laboratory for titration. Unfortunately, carbon dioxide from the air is readily absorbed by dilute caustic. To eliminate this absorbed carbon dioxide, Sterner[4] points out the necessity of rapidly heating to the boiling point the nearly neutral solution.

In order to overcome disadvantages of such methods, a sampling solution was sought which would not be affected by normal atmospheric concentrations of carbon dioxide, which would give immediate, consistent answers in the field, and which would have sufficient sensitivity to detect quantitatively concentrations as low as 0.5 ppm.

Development of Method

A SOLUTION having all these desirable properties proved to be a 1:1 mixture of glycerol and water. Carbon dioxide in air in concentrations as high as 5000 ppm will not affect this solution. Since 5000 ppm is about 10 to 15 times the normal atmospheric concentration of carbon dioxide, errors result-

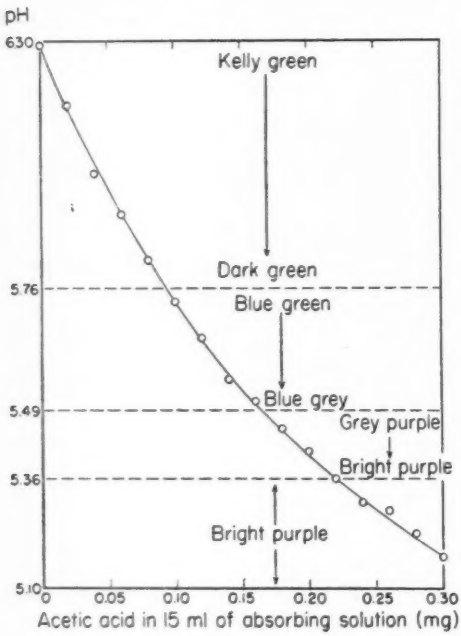

Fig. 1.
Effect of pH of 1-1 glycerol-water solution on Fleisher Methyl Purple Indicator.

ing from this source were avoided.

While the glycerol-water solution was not affected by carbon dioxide, it had a serious disadvantage because of its marked tendency to foam. Aspirating air through the solution for only a few seconds caused foam to rise into the suction line and flow meter. To reduce foaming, a few drops of octyl alcohol were at first used. Recently, the octyl alcohol has been replaced with Dow Corning Antifoam A, a commercially available silicone antifoamer which has proved much more efficient than octyl alcohol.

Next, an indicator was sought which would give an easily recognized color change in the right pH range. After a trial of nearly all common indicators, the best one for the purpose appeared to be Fleisher Methyl Purple.[5] This indicator has the following advantages:

1. Of the indicators tried, Fleisher Methyl Purple proved to be the most sensitive. The color change is from a Kelly green through gray to a definite purple within a range of one pH unit. Fig. 1 shows the color of the indicator at various pH's. This color change is easily recognized under many lighting conditions, even in the semi-darkness of photographic processing rooms. (A dim flash light with dark green filter is used when testing under such lighting conditions.)

2. Fleisher Methyl Purple is commercially available as a ready-to-use solution.

3. This indicator functions well in the sampling solution described above.

Reagents and Calibration

AS FINALLY evolved, the procedure for preparing the absorbing solution is as follows: equal volumes of glycerol (Eastman Kodak Company, Catalogue No. 339) and distilled water are mixed and two drops of Fleisher Methyl Purple indicator are added to each 10 milliliters of glycerol-water solution.

Sufficient Dow Corning Antifoam A is added to produce a noticeable surface film when viewed by reflected light. Approximately three to four drops of this viscous compound added to 500 milliliters of solution and stirred vigorously for several minutes will produce this film. Antifoam A is very effective despite its immiscibility with the absorbing solution.

The absorbing solution is calibrated as follows: Using a five milliliter microburette, a measured volume of the absorbing solution (equal to that used for the air sample) is titrated with acetic acid solution prepared by diluting 0.953 milliliters of 100% acetic acid to one liter with distilled water (concentration equals one milligram per milliliter). This titration must be repeated for each new mixture of glycerol-water absorbing solution.

Air Sampling

TO TAKE a sample, the acid contaminated air is drawn through a gas washing bottle containing a measured volume of the absorbing solution. The volume of air required to change the color of the absorbing solution from Kelly green to purple is measured with a flow meter and the air concentration of acetic acid is then calculated from the following formula:

$$\text{ppm} = \frac{T \times 14.7}{L}$$

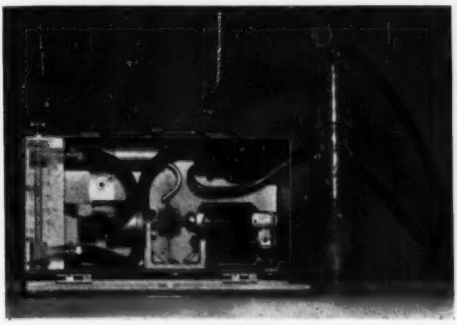

Fig. 2.

where

ppm = parts per million of acetic acid vapor in air

T = milligrams of acetic acid used in the calibrating titration

L = cubic feet of air sampled

At concentrations from 1 to 10 ppm, 15 milliliters of absorbing solution and a sampling rate between 0.1 and 0.2 cubic feet per minute were most satisfactory. For concentrations of from 10 to 20 ppm, 45 milliliters of absorbing solution and a sampling rate of 0.1 cubic foot per minute proved to be the best.

A Gast Portable Sampler coupled to a gas washing bottle is a convenient and compact sampling device. Fig. 2 shows the apparatus. The Gast Sampler was altered slightly by removing the entire air sampling head and replacing it with a trap to prevent carry-over mist and condensed vapor from reaching the flow meter or pump.

Efficiency

NO DIFFERENCE in efficiency was found between gas washing bottles equipped with fritted glass cylinders or bottles having impinger type nozzles. Absorption checks were also made using several bottles in series. Essentially complete absorption of acetic acid occurs in the first bottle in series.

Fig. 3 shows the range of recoveries for concentrations from 1 to 20 ppm. To obtain these data, air containing known concentrations of vapor between 1 and 20 ppm was aspirated through 15 milliliters of absorbing solution; the volume of air required to effect a color change was noted in each case. Flows ranging from 0.1 to 0.2 cubic feet per minute were used and no difference in absorption efficiency was noted within this range.

At concentrations above 15 ppm, there was an increased spread of the determined values around the actual values. This resulted from observer error in timing the end point.

When sampling these higher concentrations, a few seconds' error in recording the time of the end point will cause an appreciable difference in the determined concentrations. To escape this difficulty, lower flow rates and/or larger volumes of the absorbing solution are used. Very low flow rates may be achieved by using a 50 cc

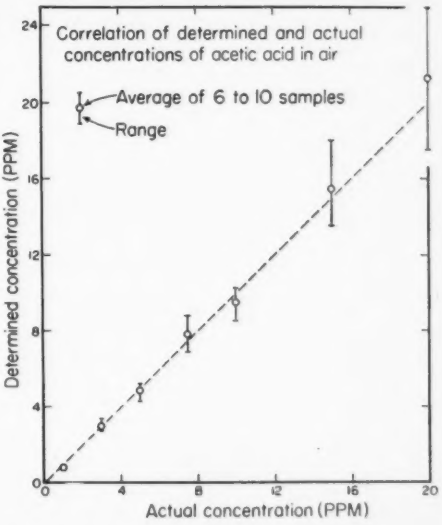

Fig. 3.

Fig. 4.

syringe (Fig. 4). This simple device has proved very useful for field sampling, particularly where concentrations above 20 ppm are encountered. Using this syringe, up to 100 ppm of acetic acid were easily determined.

Comments on Method

THIS METHOD possesses adequate sensitivity and is free from carbon dioxide interference. It should be noted that there is sufficient difference in various batches of absorbing solution to make titration of each new batch necessary. In fact, this restandardization is necessary if the glycerol-water solution is more than one week old.

When sampling low concentrations (below 5 ppm) some experience is needed to recognize the correct end point. This experience can be best achieved by sampling known acetic acid vapor air mixtures made in the laboratory. It has been found that there is a slight tendency to oversample and that the truest end point occurs when the solution first attains a definite (although faint) over-all purple color.

It is advisable to segregate glassware contaminated with Antifoam A. It is better not to wash these pieces with other laboratory ware since the antifoam clings to walls of burettes and volumetric flasks and is very difficult to remove.

Conclusion

TO DATE, the only acid contaminants sampled by the method described here have been acetic and trifluoroacetic acids. The method can possibly be adapted to the field determination of many other organic and inorganic acids.

Both static and dynamic systems have been used to prepare known concentrations of acetic acid vapor, and the results of sampling such mixtures by this method have always been well within the limits of accuracy required by the Industrial Hygienist. During the past four years, equally good results have been obtained in field sampling. Especially valuable has been the adaptability of the method to many types of sampling apparatus. The procedure described here should prove useful whenever a convenient, reasonably accurate, on-the-spot determination of acetic acid vapor concentrations is required.

References

1. PASSER, H. C.: Economic Analyst, Eastman Kodak Company, Rochester, New York. Personal communication.
2. *AMA Arch. Ind. Health*, 11:521-524, (June) 1955.
3. FEIGL, FRITZ: *Spot Tests*, Volume II, Organic Applications. Elsevier Publishing Company, New York.
4. PATTY, FRANK A., Editor: Industrial Hygiene and Toxicology, Volume II. Interscience Publishers, Inc., New York.
5. Manufactured by Fleisher Chemical Company, Benjamin Franklin Station, Washington 4, D. C. Distributor: E. Machlett and Son, New York 10, New York.

Industrial Hygiene Engineer Wanted

SAN DIEGO COUNTY has an immediate opening for an industrial hygiene engineer who will be responsible for the county public health industrial hygiene and air pollution programs. Among the duties will be making surveys, directing an analytical laboratory and providing guidance to industry, concerning hazards of materials and processes. The applicant should be 25-54 years of age and a college graduate with a major in civil, mechanical or industrial engineering. Three years of experience in public health engineering, one of which must be in the field of industrial hygiene engineering, are required. One year of graduate work in public health or industrial hygiene may be substituted for a year of experience. The salary scale is from $587.00 to $647.00 per month. There may be a written examination if enough qualified candidates apply. Application forms and further information are available from the Department of Civil Service and Personnel, Room 402 Civic Center, San Diego 1, California.

Industrial Hygiene Units in Industry

A SURVEY BY THE AIHA DEVELOPMENT COMMITTEE

A QUESTIONNAIRE designed to develop information useful to industries contemplating new or reorganized industrial hygiene units was mailed to members of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION known to be employed in key positions in industry. The questionnaire was arranged so as to avoid identity of the person or organization participating. Of the 133 questionnaires mailed out, 57 were filled in and returned, 10 were returned uncompleted, and 66 did not reply. Some returns neglected to reply to some questions. The tabulation of results is shown in the table which follows.

Of the ten questionnaires returned uncompleted, one was a duplicate mailed to a second member of a participating group, three were returned because of uncrystallized programs in the development stage, three were returned by the postal service unopened, two were returned "because of security regulations" and one because the industrial hygiene activities were rudimentary while the health physicist in charge was of the opinion that the program was completely satisfactory to management. A number of questionnaires were not returned because more than one had gone to certain units and also because several industrial members of AIHA do not have active industrial hygiene programs.

The number of employees per member of industrial hygiene staff varies widely with no definite central tendency. However, about 53% lie between 4,000 and 15,000. In general, the larger industries have more employees per industrial hygiene member than do the smaller industries. Several of the industries with less than 5,000 employees per member of the industrial hygiene staff are engaged in manufacturing chemicals and drugs and are conducting toxicological research. Several in the group above 20,000 plan to add to industrial hygiene personnel.

The activities in which these 57 units are engaged are most interesting. Several units engage in all of the activities named, while others are active in only a few. A rather surprising number are conducting efficiency tests of air cleaners as well as air pollution work of various sorts. The time spent on air pollution work for the majority of those con-

TABULATION OF SIGNIFICANT INFORMATION
REGARDING INDUSTRIAL HYGIENE UNITS IN INDUSTRY

Items	Number of Individual Companies Classified by Type of Industry							
	Total All Types	Chemical and Drugs	Miscellaneous Manufacturing	Petroleum and its Products	Steel	Mining Smelting and Refining	Manufacturers of Electrical Equipment	A. E. C. Projects
Questionnaires mailed	133							
Questionnaires completed and returned	57	17	13	8	7	5	4	3
Questionnaires returned uncompleted	10	2	2	2	1	1		2
Number of employees per member Industrial Hygiene Staff								
150-400	3							3
700-1,000	2					1	1	
2,000-3,000	8	5	2			1		
4,000-5,000	14	3	5	4	1	1		
6,000-9,000	6	1	1	1	3			
10,000-15,000	10	3	2	1	1		3	
16,000-20,000	5	2	2			1		
30,000-35,000	3	3	1	2	2			
Activity Initiated -								
1920-1929	1						1	
1930-1939	15	3	7	1	1	2	1	
1940-1949	26	11	5	4	4	2		
1950-1955	13	3	1	3	2		2	2

TABULATION OF SIGNIFICANT INFORMATION
REGARDING INDUSTRIAL HYGIENE UNITS IN INDUSTRY—CONTINUED

Items	Number of Individual Companies Classified by Type of Industry							
	Total All Types	Chemical and Drugs	Miscellaneous Manufacturing	Petroleum and its Products	Steel	Mining Smelting and Refining	Manufacturers of Electrical Equipment	A. E. C. Projects
Analytical Work Conducted by Field Personnel—								
None	13	3		2	2	2	2	2
<10%	15	5	3	3	3			1
10% to <50%	5		2	2			1	
50% to <100%	6	3	3					
100%	9	3	3		2		1	
Industrial Hygiene Personnel Do all Lab Work—								
Yes	23	3	9	3	4	2	2	
No	31	13	4	5	3	1	2	3
Lab Used Solely for Industrial Hygiene—								
Yes	29	4	9	5	4	3	2	2
No	23	11	4	3	3		2	
Total Lab and Office Space, Square feet per person—								
Range	70-1100	100-400	125-250	180-350	70-1100	350-420	100-300	107-133
Average	245	191	179	250	383	390	200	120
One Industrial Hygiene Unit Services all Plants—								
Yes	46	14	12	6	6	4	2	2
No	6	3			1		2	
Reports "Approved" by other than Industrial Hygiene Personnel—								
Yes	21	6	4	6	3	1	1	
No	35	10	9	2	4	4	3	3
Compliance with Industrial Hygiene Recommendations—								
Optional	42	11	9	8	5	4	4	1
Obligatory	13	5	4		2			2
Analytical Instruments Available in Industrial Hygiene Lab—								
Mass Spectrometer	0							
Emission Spectroscope	3		1	1	1			
X-Ray Diffraction Unit	4			1	3			
Spectrophotometer								
(visible light)	23	5	6	3	4	1	2	2
(ultraviolet)	11	3	1	1	3		1	2
(infrared)	5			1	3			1
(fluorescent)	12	2	2	2	2	1	1	2
Ionizing Radiation Survey Meters	29	9	7	5	5		1	2
Equipment Available but not in Industrial Hygiene Lab—								
Mass Spectrometer	34	14	6	5	4		3	2
Emission Spectroscope	30	10	7	5	2	1	3	2
X-Ray Diffraction Unit	35	15	8	5	2	1	2	2
Spectrophotometer								
(visible light)	18	7	4	3	2	1	1	
(ultraviolet)	26	10	8	4	2	1	1	
(infrared)	30	13	8	4	2	1	1	1
(fluorescent)	16	7	3	3	2	1		
Ionizing Radiation Survey Meters	16	6	3	2		1	3	1
Present Arrangement Satisfactory?								
Yes	42	13	13	6	3	3	1	3
Qualified Yes	3	2			1			
Suggested Improvement	15	4	1	2	1	1	4	2
No	2	1					1	

TABULATION OF SIGNIFICANT INFORMATION
REGARDING INDUSTRIAL HYGIENE UNITS IN INDUSTRY—CONTINUED

Items	Number of Individual Companies Classified by Type of Industry							
	Total All Types	Chemical and Drugs	Miscellaneous Manufacturing	Petroleum and its Products	Steel	Mining Smelting and Refining	Manufacturers of Electrical Equipment	A. E. C. Projects
Activities								
Appraise Environment for—								
Air Contaminants	56	17	12	8	7	5	4	3
Heat and Humidity	51	15	11	8	6	5	4	2
Noise	48	16	11	8	5	2	4	2
Radiant Heat	44	10	10	8	6	4	4	2
Illumination	42	11	10	7	5	4	3	2
Ionizing Radiation	42	14	10	8	5		3	2
Ultraviolet Radiation	36	13	7	6	5	1	4	
Deal with Air-Borne Waste	41	12	10	5	4	4	4	2
Analyze Biological Specimens	33	7	8	7	4	3	2	2
Make Clinical Tests	31	6	9	5	3	2	3	3
Efficiency Test of Air Cleaners	29	7	9	5	2	5		1
Deal with Liquid Wastes	19	5	5	3	1	2	1	2
Make Specific or Detailed Recommendations for Control of Unsatisfactory Conditions	50	13	12	7	7	4	4	3
Detailed Recommendations for—								
Process Ventilation	35	8	10	6	5	3	2	1
General Ventilation	32	5	9	6	5	3	1	3
Make-up Air Supply	31	7	8	6	5	3	1	1
Top Management Representative "carrying the ball" for Industrial Hygiene								
Vice-President or Representative, Director of Personnel, Personnel Services or Industrial Relations.	23	6	6	4	2	2	2	1
Vice-President in Production or Works Manager	12	5	3	1	1	2		
President, Chairman of Board, or Board of Directors	6	1	2	2	1			
Medical Director	6	2	1		2			1
Superintendent of Production	2			1		1		
Superintendent of Central Engineering	2	1	1					
Vice-President of Research	1		1					
Manager of Industrial Hygiene	1	1						
Manager of Safety	1	1						
Legal Staff	1				1			
Construction Superintendent	1							1
Under whom does Industrial Hygiene Director Work—								
Medical Director	31	11	4	7	6	1		2
Director of Personnel, Personnel Service or Industrial Relations	6		4			1	1	
Manufacturing Manager	3	1				2		
Manager Safety and Industrial Hygiene	2		1				1	
Supervisor Special Health Services	2		1			1		
Chief Chemist	2				1		1	
President	1		1					
Vice-President in charge of Production	1	1						
Vice-President of Research	1		1					
Director Chemical Research	1	1						
Director Biochemical Research	1	1						
Chief Engineering Department	1		1					
Superintendent of Construction	1							1
Safety Manager	1	1						
Central Service Engineer	1	1						
Chief Sanitary Engineer	1			1				
Supervisor on Manufacturing Staff	1						1	

cerned amounts to less than 10% but ranges from less than 1% to 95%. The replies concerning biological analyses and clinical tests were apparently confused by some misinterpretations of "clinical" tests. They are believed to be inaccurate. The chemical and drug group appear to participate less than the average in making detailed recommendations for ventilation.

About 40% of top management's interest in industrial hygiene centers in the personnel and industrial relations group. Production is second with 24%. It is noteworthy that in about 10% of the industries, management's interest is concentrated in the Board of Directors or the President, while in another 10%, the Medical Director is the principal interested management representative. The man under whom the industrial hygienist works is, in 54% of the industries, the Medical Director; 10% are directed by the Director of Personnel, personnel services or industrial relations; another 5% are directed by the Manufacturing Manager; but the many different ways in which the industrial hygiene activity has been set up in the industrial organization are striking. The position of the chief industrial hygienist on the organization chart ranges from the sixth level up to the second level. His position in the organization is apparently influenced by many things, including the type and size of the industry and the time the activity was initiated. Each of the sixth level locations for industrial hygiene is in a large company in which the activity dates back more than 20 years. It is not surprising that replies from the people in these units indicate a certain amount of frustration because of inability to accomplish the objectives that are possible in industrial hygiene. It is of interest that 70% of industrial hygiene units in industry have been initiated since 1940.

Another item of interest is that in over 15% of these industrial hygiene units the field men do all the analytical work also, while in about 50% of the units the field personnel do less than 10% of the analytical work. These differences do not appear to correlate with the size of the organization or other available information.

The total space devoted to industrial hygiene for office and laboratory averages around 245 square feet of floor space per person, but the range is 70 to 1,000.

In 30% of the cases the industrial hygiene report is "approved" by other than industrial hygiene personnel before it is sent to the departments concerned. Since over 50% of the industrial hygiene units are directed by medical personnel a significant number of these directors apparently do not choose to judge and rule over the contents of the report, but prefer to leave this responsibility with the industrial hygiene unit preparing the report. Whether the recommendations are mandatory (23%) or advisory does not appear to be related to the size of the industry, the relation of the medical director to the industrial hygiene activity, nor to the age of the unit.

The availability of major expense analytical instruments is not surprising. Specialists are required to operate much of this equipment and when it is located in another department it can more readily be applied to problems outside industrial hygiene.

Perhaps the outstanding fact brought out by the survey is that 45 of the 47 persons responding to the question of whether the set-up is workable and desirable are satisfied with their industrial hygiene unit and the way in which it operates. The most significant suggestions for improvement center around the problem of having only a liaison with plant engineering personnel. There is a considerable strain placed on this tie when the plant engineering personnel must call upon the Medical Director for any engineering help they wish from industrial hygiene. This is especially so when the report is in turn forwarded by the Medical Director. Other suggestions included a need for more toxicological research among the chemical and drug group, and more personnel in all groups. One reply stated that in the interest of efficiency and reduction of waste, health physics should be combined with industrial hygiene and should, along with safety, report to the same director of administrative functions rather than operation functions. Other suggestions stemming from units finding themselves in the sixth level of the organization express need for reorganization and centralization of industrial hygiene activities, and a greater need of interest from top management.

—AIHA DEVELOPMENT COMMITTEE: F. A. Patty, Chairman; W. G. Hazard; J. C. Radcliffe; J. C. Soet; A. J. Vorwald, M.D.

HYGIENIC GUIDE SERIES

FLUORINE

I. Hygienic Standards

A. RECOMMENDED MAXIMUM ATMOSPHERIC CONCENTRATION (8 hours): 0.1 parts of gas per million parts of air, by volume (ppm).[1]

(1) *Basis for Recommendation*: Toxicologic observations on animals.[3]

B. SEVERITY OF HAZARDS:

(1) *Health*: Extra hazardous for acute; high, for chronic.

(2) *Fire*: Under suitable conditions, fluorine reacts spontaneously at room temperature with most materials except inert gases, metal fluorides in their highest valence states, and carbon tetrachloride. With metals it reacts only slowly, but under some conditions of elevated temperature, fluorine at atmospheric pressure can burn steel equipment. For reasons unknown, fluorine does not always react with water, but at times reaction may occur with explosive violence.

C. SHORT EXPOSURE TOLERANCE: Moderate to severe pulmonary irritation occurs at all concentrations down to three milligrams of gas per cubic meter of air (1.9 ppm). The tolerated exposure was taken as 1.7 milligrams per cubic meter of air (1 ppm) in experiments of Stokinger.[3]

D. ATMOSPHERIC CONCENTRATION IMMEDIATELY HAZARDOUS TO LIFE: Fluorine was uniformly fatal to rabbits, guinea pigs, rats and mice in exposures ranging from five minutes at 10,000 ppm to three hours at 200 ppm.[3] Exposure to fluorine under 40 pounds of pressure at a distance of one inch for 0.6 seconds led to flash burns resembling those induced by an oxyacetylene flame.

II. Significant Properties

A yellow, highly reactive, gas. It can be piped through standard steel pipe or copper tubing, equipped with Monel metal or nickel valves (Teflon packing). With the moisture of the air, it forms hydrogen fluoride and, possibly, oxygen fluoride, OF_2,[4] a compound more toxic than fluorine.

Chemical formula:	F_2
Molecular weight:	38.00
Specific gravity:	1.108 at -187°C
Boiling point:	-187°C
Relative vapor density:	1.31 (air = 1)
At 25°C and 760 mm Hg,	
1 ppm of vapor:	0.00155 mg/liter
1 mg/liter:	643 ppm
Solubility:	Reacts with water and most other solvents.

III. Industrial Hygiene Practice

A. RECOGNITION: Used in the synthetic chemical industry.

B. EVALUATION OF EXPOSURE: No reliable methods available. In animal experimental work, reliance had to be placed on the results of exposure to metered dilutions of fluorine in nitrogen.[3]

C. RECOMMENDED CONTROL PROCEDURES: Special precautions required in the handling of this gas have been described in detail by Landau and Rosen[2] and must be rigidly enforced. The design and housing of equipment must be entrusted only to thoroughly trained and experienced personnel.

IV. Specific Procedures

A. FIRST AID: Too few accidents have been reported for new specific procedures to be evaluated. Remove the individual immediately to an uncontaminated area and administer 100% oxygen intermittently, in cases of respiratory involvement. Enforce bed rest for at least 24 hours, observing for any symptoms of pulmonary involvement. For cutaneous contact, flush the area with copious amounts of water, followed by swabbing with a 2.8% solution of aqueous am-

monia. Immerse affected part in a bath of water for a prolonged period. Injection of the tissue with 10% calcium gluconate around the affected area is helpful. Maintain workroom atmospheres at all times below 0.1 ppm. This will require careful process enclosure and the use of mechanical ventilation.

B. BIOCHEMICAL ASSAY: No methods available.

C. SPECIAL MEDICAL PROCEDURES (including preplacement): Persons showing any evidence of respiratory abnormality should be restricted from fluorine exposure.

V. Literature References

1. American Conference of Governmental Industrial Hygienists: *AMA Arch. Ind. Health*, 11:521, 1955.
2. LANDAU, R., and ROSEN, R.: *Ind. Eng. Chem.*, 39:281, 1947.
3. STOKINGER, H. E., *et al*: Natl. Nuclear Energy Ser., Manhattan Project Tech. Sect., Div. VI, 1. Pt. II, 1021-57, 1949.
4. Symposium on Fluorine Chemistry: *Ind. Eng. Chem.*, 39:236, 1947.

FLUORIDE-BEARING DUSTS AND FUMES

I. Hygienic Standards

A. RECOMMENDED MAXIMUM ATMOSPHERIC CONCENTRATION (8 hours): 2.5 mg (calculated as fluoride ion) per cubic meter of air (mg/m^3).[1]

(1) *Basis for Recommendation*: Nasal irritation in humans in the steel producing and magnesium foundry industries, and upon the mean urinary fluoride levels of exposed workers in the electrolytic aluminum and fertilizer manufacturing industries.

B. SEVERITY OF HAZARDS:

(1) *Health*: Moderate for acute exposures, high for chronic. After years of exposure only a very few workers in this country have given radiographic evidence of fluoride retention in their bones. In Danish workers exposed to cryolite dust, instances of pronounced skeletal abnormalities have been recorded. Fluoride-bearing fumes and dusts of particles five microns or less in size are readily absorbed when inhaled and promptly result in an increase in the urinary fluoride excretion. When excessive amounts are inhaled, this excretion lags behind the daily intake[3] and fluoride retention occurs, particularly in the bones. If this storage continues over a sufficiently long period, the bones may show an increased radiographic density, and eventually structural abnormalities may develop.

(2) *Fire*: None.

C. SHORT EXPOSURE TOLERANCE: Tolerance varies widely, depending upon compound.

D. ATMOSPHERIC CONCENTRATION IMMEDIATELY HAZARDOUS TO LIFE: Unknown.

II. Significant Properties

Properties dependent upon specific compound.

III. Industrial Hygiene Practice

A. RECOGNITION: The use of fluoride-coated welding rods offers a definite hazard.[6] In magnesium foundries, fluoride concentrations in excess of the recommended maximum atmospheric concentration have been recorded, particularly in the core-spraying and pouring areas.[8] Atmospheric pollution in the vicinity of aluminum refining and rock phosphate treatment plants may be a serious problem,[7] particularly with respect to vegetation and livestock damage.

B. EVALUATION OF EXPOSURE:

(1) *Instrumentation*: No direct reading instruments available.

(2) *Chemical Method*: The dust may be collected by any of the standard techniques, involving filtration, electrostatic precipitation, or impingement. Determination of the fluoride ion by the method of Dahle, Bonnar, and Wichmann[5] is subsequently made, after the addition of calcium or magnesium oxide and distillation with perchloric acid.

C. RECOMMENDED CONTROL PROCEDURES: Maintain workroom atmospheres below 2.5 mg/m^3. This requires good enclosure of processes and, usually, some process ventilation.

IV. Specific Procedures

A. FIRST AID: None.

B. BIOCHEMICAL ASSAY: Urinary fluoride data are of great value when applied to a group of exposed personnel. However, data on single individuals are of questionable value, because of inability to correlate exposure with excretion data.[4] Levels above 4 mg of fluoride ion per liter of urine are indicative of excessive exposure. For the determination, a composited sample, prepared from spot samples collected on five separate days, should be used.[2]

C. SPECIAL MEDICAL PROCEDURES: Radiographic surveys of all exposed personnel should be made at intervals of a few years. Chest films, when a part of periodic physical examination, can be used in this respect. Supplemental films of the pelvis or lower back can be made in those cases where increased bony density is suspected. The interpretation of the roentgenograms should be made by radiologists with special experience in this field.

V. Literature References

1. American Conference of Governmental Industrial Hygienists: *AMA Arch. Ind. Health*, 11:522, 1955.
2. COLLINGS, JR., G. H. (Army Environmental Health Laboratory, Edgewood, Md.): Industrial Health Surveys (In Press).
3. COLLINGS, JR., G. H., *et al*: *Arch. Ind. Hyg. & Occup. Med.*, 4:585, 1951; 6:368, 1952.
4. CRALLEY, L. V. (Aluminum Company of America): Personal Communication.
5. DAHLE, DAN; BONNAR, R. U., and WICHMANN, H. J.: *J. Assoc. Official Agr. Chem.*, 21:459, 1938.
6. DRINKER, P., and NELSON, K. W.: *Ind. Med.*, 13:673, 1944.
7. Fluorosis Committee of the Medical Research Council (London): Memorandum No. 22, 1949.
8. LARGENT, E. J., and FERNEAU, I.: *J. Ind. Hyg. & Tox.*, 26:113, 1944.

ARSINE

I. Hygienic Standards

A. RECOMMENDED MAXIMUM ATMOSPHERIC CONCENTRATION (8 hours): 0.05 parts of vapor per million parts of air, by volume (ppm).[1]

(1) *Basis for Recommendation*: Human experience and experimental animal studies.

B. SEVERITY OF HAZARDS:

(1) *Health*: Extra hazardous for acute and chronic exposures. May cause serious or fatal destruction of red blood cells and jaundice on a single short exposure.

(2) *Fire*: Practically none.

C. SHORT EXPOSURE TOLERANCE: Uncertain and probably variable. One to ten parts per million for an hour probably dangerous.[4,5]

D. ATMOSPHERIC CONCENTRATION IMMEDIATELY HAZARDOUS TO LIFE: Uncertain but probably greater than 100-200 parts per million.

II. Significant Properties

A colorless gas with slight garlic-like odor.

Chemical formula:	AsH_3
Molecular weight:	77.9
Boiling Point:	-55°C
Relative vapor density:	2.7 (air = 1)
Solubility:	20 volumes in 100 volumes of water (20°C)

At 25°C and 760 mm,

1 ppm:	0.0032 mg/l
1 mg/liter:	313 ppm

III. Industrial Hygiene Practice

A. RECOGNITION: Arsine may be generated in any pickling operation when the metal contains arsenic. It also arises from certain metal drossing operations. Symptoms are usually delayed, with the passage of dark red urine four to six hours or more after exposure (the first indication), followed by abdominal or back pain and cessation of urination.

B. EVALUATION OF EXPOSURE:

(1) *Instrumentation*: No direct reading instruments available.

(2) *Chemical Method*: By means of mercuric bromide test papers through which a known volume of air is drawn,[2] by the Gutzeit[6] method, after collecting the gas in water in an all-glass device, or by the method of Jacobs and Nagler.[3]

C. RECOMMENDED CONTROL PROCEDURES: Maintain workroom atmospheric concentrations below 0.05 ppm through the use of process ventilation and enclosure. Store metals and metal dross in dry areas.

IV. Specific Procedures

A. FIRST AID: Put at bed rest and obtain medical care as soon as there is any knowledge of exposure. Any worker known to have been exposed to a high concentration should be hospitalized at once. There are no specific first aid measures, other than immediate removal from exposure.

B. BIOCHEMICAL ASSAY: Arsenic in the urine can be determined as outlined by Elkins.[2] Urinary levels above 0.5 mg./liter are indicative of harmful exposure.

C. SPECIFIC MEDICAL PROCEDURES: Obtain blood type and donors. Measure intake and output of urine. Treat as in other types of acute hemolytic anemia. Give replacement transfusions as necessary. BAL is *ineffective* in arsine poisoning. The prognosis after a severe exposure is bad and the mortality high.

V. Literature

1. American Conference of Governmental Industrial Hygienists: *AMA Arch. Ind. Health*, 11:522, 1955.
2. ELKINS, H. B.: Chemistry of Industrial Toxicology. John Wiley and Sons, Inc., New York, 1950.
3. JACOBS, M. B., and NAGLER, J.: *Ind. and Eng. Chem.* (Anal. Ed.) 14:442, 1942.
4. MORSE, K. B., and SETTERLIND, A. N.: *Arch. Ind. Hyg. & Occup. Med.* 2:148, 1950.
5. PATTY, F. A. (Editor): Industrial Hygiene and Toxicology, Volume 2. Interscience Publishers, New York and London, 1949.
6. SNELL, F. D., and SNELL, C. T.: Colorimetric Methods of Analysis. D. VanNostrand Co., Inc., New York and London, 1949.

NITROGEN DIOXIDE

I. Hygienic Standards

A. RECOMMENDED MAXIMUM ATMOSPHERIC CONCENTRATION (8 hours): 5 parts of gas per million parts of air, by volume (ppm).[2]

(1) *Basis for Recommendation*: Extensive animal studies and human experience in industry.[1,5,7]

B. SEVERITY OF HAZARDS:

(1) *Health*: High for both acute and chronic exposures. Exposures of relatively short duration to concentrations above 5 ppm produce cough and irritation of the respiratory tract. Continued exposures to concentrations much above 5 ppm may produce a slowly progressive and often fatal pulmonary edema and hemorrhage. High concentrations irritate mucous membranes and wet skin surfaces.

(2) *Fire*: Ordinarily none, but may be flammable in presence of pure oxygen. Nitrated products may present a fire hazard.

C. SHORT EXPOSURE TOLERANCE: Safe level unknown, but 25 ppm probably safe for a few minutes.

D. ATMOSPHERIC CONCENTRATION IMMEDIATELY HAZARDOUS TO LIFE: 200 to 700 ppm is rapidly fatal.[6]

II. Significant Properties

A reddish brown gas above 21.3°C; a nearly colorless liquid between 21.3°C and -9.3°C; a white solid below -9.3°C. At 40°C, approximately 30% is NO_2 and 70% N_2O_4.

Chemical formula:	NO_2
Molecular weight:	46.01 (100% NO_2 assumed)
Specific gravity:	1.448 (20°C)
Boiling point:	21.3°C
Relative vapor density:	1.59 (air = 1)
At 25°C and 760 mm Hg,	
1 ppm of vapor:	0.00188 mg/liter
1 mg/liter:	532 ppm
Odor:	Pungent, sweetish. Threshold of detection, about 5 ppm
Visual threshold:	75-150 ppm
Solubility:	In water and most organic solvents.

III. Industrial Hygiene Practice

A. RECOGNITION: May be found around nitric acid dip tanks, some JATO propellants, welding fumes, cellulose nitrating, decomposition of nitrated materials, diazotization processes, blasting, and automobile exhausts. In most industrial applications, other oxides of nitrogen may exist. However, they are of substantially less significance for industrial health than nitrogen dioxide.

B. EVALUATION OF EXPOSURES: Sampling and analysis of atmospheric concentrations may be done by either the method of Patty[8]

or of Saltzman.[9] Methods consist of absorbing gas in reagent and measuring intensity of color developed. A commercial field kit utilizing the method of Patty[8] is available from Mine Safety Appliances Company, Pittsburgh.

C. RECOMMENDED CONTROL PROCEDURES: Maintain workroom concentrations below 5 ppm. This will usually require process ventilation and enclosure. All potentially exposed personnel must be trained in the recognition of low concentrations of nitrogen dioxide and in the use of protective and first aid equipment. Safety showers and eye washing fountains should always be available around nitration operations.

IV. Specific Procedures

A. FIRST AID: With any obvious exposure, remove individual immediately from contaminated atmosphere. Call a physician and advise him of the type of exposure. Keep at complete rest until seen by a physician. For eye or skin contact, wash with copious amounts of water and refer to a physician.

B. BIOCHEMICAL ASSAY: Usually not, but methemoglobin determinations may be of value.

C. SPECIAL MEDICAL PROCEDURES:

(1) *Preplacement*: Individuals with pre-existing pulmonary disease should not be exposed.

(2) *Treatment of acute exposures*: Give patient immediate and complete bed rest. Immediate medical examination to evaluate extent of pulmonary damage is a "must." If early transient asthmatic breathing is present, pulmonary edema may later occur. Such patients must be kept under constant medical supervision for at least 24 hours. The blood pressure, pulse, and respiratory rates should be noted at frequent intervals. Total white and differential count, and platelet count may be useful in predicting pulmonary damage.[3,4] 100% oxygen should be used as indicated. If no history of coughing, difficulty of breathing, or weakness develops, and if the above tests are normal at the end of eight hours, pulmonary edema will probably not occur.

(3) *Periodic medical examinations*: Individuals who are repeatedly exposed to low concentrations should have routine medical examinations, including chest x-rays. Those who have experienced acute exposures should be followed with medical examinations for some time.

V. Literature References

1. ADLEY, F. E.: *J. Ind. Hyg. & Tox.*, 28:17, 1946.
2. American Conference of Governmental Industrial Hygienists: *AMA Arch. Ind. Health*, 11:522, 1955.
3. FASSETT, D. W. (Eastman Kodak Co.): Personal Communication.
4. FLEMING, ALAN: *Ind. Med. & Surg.*, 12:127, 1943.
5. GRAY, E. LEB., *et al*: *Arch. Ind. Hyg. & Occup. Med.*, 10:409-423, 1954.
6. HENDERSON, Y., and HAGARD, H. W.: Noxious Gases. Rheinhold Publishing Company, New York, 1943.
7. LINDQUIST, T.: *Acta Med. Scand.*, 118:210, 1944.
8. PATTY, F. A., and PETTY, G. M.: *J. Ind. Hyg. & Tox.*, 25:361, 1943.
9. SALTZMAN, B. E.: *Anal. Chem.*, 26:1949, 1954.

CHROMIC ACID

I. Hygienic Standards

A. RECOMMENDED MAXIMUM ATMOSPHERIC CONCENTRATION (8 hours): 0.1 milligram of chromic acid per cubic meter of air (mg/m^3).[2,3]

(1) *Basis for Recommendation*: Human experience in industry,[5,6] but limited to exposures above 0.1 mg/m^3.

B. SEVERITY OF HAZARDS:

(1) *Health*: Moderate for chronic exposure; low, for acute. Chromic acid has a powerful caustic action on mucous membrane, conjunctiva and skin, producing ulcers known as "chrome holes." These are found frequently on the hands and are of most concern when they occur in the nose as a result of breathing chromic acid mist produced by electroplating baths. Ulcers in the nose may ultimately result in a perforated septum which does not, however, impair nasal function nor constitute a disabling condition. The ulcers

are fairly painless and temporary disability, when it occurs, is short. Permanent disability is unknown although some states have a disfigurement statute and loss of nasal septa is considered a form of permanent disability. Allergic reactions have been noted but are believed to be rare.

(2) *Fire*: None.

C. SHORT EXPOSURE TOLERANCE: Unknown.

D. ATMOSPHERIC CONCENTRATION IMMEDIATELY HAZARDOUS TO LIFE: Unknown.

II. Significant Properties

Chromic anhydride (CrO_3) is often referred to as chromic acid. It forms chromic (H_2CrO_4) and dichromic ($H_2Cr_2O_7$) acids when dissolved in water. Chromic acid solutions are often prepared by dissolving sodium dichromate in sulfuric acid.

Chemical formula:	CrO_3
Molecular weight:	100
Specific gravity:	2.7
Solubility:	166 grams per 100 grams of H_2O at 150°C; soluble in H_2SO_4.

III. Industrial Hygiene Practice

A. RECOGNITION: Chromic acid is extensively used for chromium electroplating and anodizing baths, in photography and other reproduction processes. Gases released at the electrodes or by aeration of anodizing tanks carry a fine mist of acid into the surrounding air where it may be inhaled by workmen. Spills, splashes and chromic acid mist affect exposed hands, arms, eyelids, etc. Plating strength is 20-25% CrO_3 with 1:200 H_2SO_4. Anodizing solutions contain 5% CrO_3. The handling of dry chromium compounds may also be hazardous.

B. EVALUATION OF EXPOSURE:

(1) *Chemical Method*: Collection by impinger, sintered absorber, or filter paper with subsequent development of a violet color with s-diphenylcarbazide.[1,9]

(2) *Test Paper Method*: Filter papers impregnated with s-diphenyl carbazide reagent can be used to give an immediate quantitative determination in the field.[11]

C. RECOMMENDED CONTROL PROCEDURES: Control of chromic acid mist from plating, anodizing and dip tanks by exhaust ventilation has been generally accepted and slot exhaust systems have been extensively developed for this application.[3,4,7,10] The addition of plastic bubbles, foams and surface tension lowering chemicals to the top of the solution has been suggested[8,12] as a substitute for part or all of the ventilation requirement. Skin protection from splashes and spills may be obtained with rubber gloves, aprons, etc. and chemical-type goggles. Showers and change of clothing should follow the day's work. Nasal mucosa should be coated with oil or vaseline prior to work. Emergency eye washing fountains should be available in the immediate working area.

IV. Specific Procedures

A. FIRST AID: Thorough washing of all affected parts with water and application of soothing ointments or oils.

B. BIOCHEMICAL ASSAY: None.

C. SPECIAL MEDICAL PROCEDURES:

(1) *Preplacement*: None.

(2) *Periodic Examination*: A physical examination of the nasal tissues and skin for the presence of "chrome holes" or ulcers should be practiced routinely. Frequency of examination should be governed by the severity of exposure.

NOTE: DR. LESLIE SILVERMAN (Harvard University) assisted the Hygienic Guides Committee in the preparation of this Guide.

V. Literature References

1. AKATSUKA, K., and FAIRHALL, L. T.: *J. Ind. Hyg. & Tox.*, 16:1, 1934.

2. American Conference of Governmental Industrial Hygienists: *AMA Arch. Ind. Health*, 11:522, 1955.

3. American Standards Association: American Standard Z37.7-1943, New York.

4. American Standards Association: American Standard Z9.1-1951, New York.

5. BLAIR, J.: *Ohio State Med. J.*, 27:142, 1931.

6. BLOOMFIELD, J. J., and BLUM, W.: *U. S. Public Health Rep.* 43:2330, 1928.

7. BRANDT, A. D.: *Heat. Pip. & Air Cond.*, 13:434, 1941.

8. HAMA, G. *et al*: *AIHA Quart.*, 15:211, 1954.

9. SALTZMAN, B. E.: *Anal. Chem.*, 24:1016, 1952.

10. SILVERMAN, L.: *J. Ind. Hyg. & Tox.*, 23:187, 1941.

11. SILVERMAN, L., and EGE, J. F., JR.: *J. Ind. Hyg. & Tox.*, 29:136, 1947.

12. SILVERMAN, L., and THOMPSON, R. M.: *J. Ind. Hyg. & Tox.*, 30:303, 1948.

Because of space limitations, it is impossible to list all methods of exposure evaluation. The selections have been made on the basis of current usage, reliability, and applicability to the usual industrial type of exposure. Any specific evaluation and/or control problem will involve professional judgment. This can best be done by professional industrial hygiene personnel.

Respiratory protective devices are commercially available. Their use, however, should be confined to emergency or intermittent exposures and not relied upon as primary means of hazard control.

A relative scale is used for rating the severity of hazards: nil, low, moderate, high, and extra hazardous.

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INDIVIDUAL Hygienic Guides in loose-leaf form may be obtained from the American Industrial Hygiene Association, 14125 Prevost, Detroit 27, Michigan, at 25c each. Also available are flexible loose-leaf binders for the loose Hygienic Guide sheets. The binders have been especially designed to provide maximum protection and ease of handling of the Guides. They will be especially useful in keeping a permanent file. The binders are blue in color, with white lettering, and are fitted with ¾" rings. The price is $1.25. The Guides and the binders may be ordered on the following order blank.

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◆ *President's Page*

THE AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, founded in 1939, has as one of its prime objectives "The promotion of the study and control of environmental factors affecting the health and well-being of industrial workers." Thus, though the members of our association represent a broad spectrum of industrial, governmental and educational groups, the focal point is always the worker.

Great progress has been made in the last 17 years in attaining the above objective. More recently, technical committees have been organized to bring together information within special fields which will be useful in studying and controlling industrial health hazards. This program will lead to the publication of technical reports or manuals in the fields of air pollution, analytical chemistry, engineering, noise, radiation and toxicology, as well as the continuation of the Hygienic Guides Series, 20 of which are already available. At the same time, we have not lost sight of the need to keep abreast of current technical developments. The papers presented at our Philadelphia annual meeting give ample evidence of the accomplishments in this phase of the ASSOCIATION'S activities.

It is only natural, therefore, that the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, which has as its main purpose the task of helping industry provide satisfactory working conditions, should take the step to make industry an integral part of its structure. Thus, it is with pride and pleasure that AIHA will in the near future extend to industry an invitation to consider the benefits arising from Industrial Associate Membership.

Lester V. Cralley

♦ Selected Titles and Abstracts

—FROM FOREIGN EXCHANGE JOURNALS

THE following is a partial list of articles, by titles and authors, from journals received by the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION since the December 1955 issue of the QUARTERLY in exchange for copies of the QUARTERLY.

Additional information on any of the journals or articles may be obtained from: CARROL S. WEIL, Senior Industrial Fellow, Mellon Institute of Industrial Research, 4400 Fifth Avenue, Pittsburgh 13, Pennsylvania.

1. THE JOURNAL OF SCIENCE OF LABOUR (JAPAN). Vol. 31, No. 9 (1955).

Discussions on Cardio-Pulmonary Function Tests of Silicotics, Especially on their Reliability Under Limited Conditions. Yutaka Sawada, pp. 589 to 596.

Changes of Physical Exercise—On Maximal Blood Pressure in Anthracosilicosis. Hirosi Yosikawa, pp. 597 to 605.

Studies on Detoxicating Factors in Liver Concentrate against Lead Poisoning: II. Comparison of Effect of Two Fractions of Liver Concentrate. Takahide Kuwaki, pp. 621 to 626.

On the Amount of Chlorine in Blood and Urine of Aluminum-Alloy Molding Plant Workers With Chronic Chlorine Gas Poisoning. Keijiro Nishimura, pp. 627 to 630.

Calculation of Permissible Time of Exposure to CO. Naosuke Sasaki, pp. 631 to 637.

II. THE JOURNAL OF SCIENCE OF LABOUR (JAPAN). Vol. 31, No. 10 (1955).

The Studies of SO_2 Poisoning: Part I. The Influence of SO_2 on the Peripheral Hemogram. Mamoru Hayashi, pp. 641 to 650.

Studies on Benzene Poisoning: Part 5. Experimental Studies on Exhalatory Elimination of Subcutaneously Injected Benzene in Rabbits. Jun Yoshioka, pp. 651 to 659.

Experimental Studies on the Toxicity of Benzene Hexachloride (Rept. 3): *IV. Effects of Lindane Vapor to Insects and Mammals.* Mitsuru Shirakawa, pp. 660 to 673.

A New Type of Sound-Guard Which Closes the External Auditory Canal by Pressing the Tragus. K. Dohi, S. Haraguchi, J. Murashima and A. Nagomitsu, pp. 695 to 697.

III. THE JOURNAL OF SCIENCE OF LABOUR (JAPAN). Vol. 31, No. 11 (1955).

Studies on Pneumoconiosis in the Earliest Stage. Akira Takanashi, pp. 727 to 741.

Studies on Benzene Poisoning: Part 6. On the Morphological Changes of Blood and Biochemical Changes of Urine in Rabbits Subcutaneously Injected with Benzene. Jun Yoshioka, pp. 773 to 784.

IV. THE JOURNAL OF SCIENCE OF LABOUR (JAPAN). Vol. 31, No. 12 (1955).

On the Change of Auditory Sensitivity Following Exposure to Loud Noises (Rept. 3): Hearing Loss and Perception of Loudness. Mitsuo Morioka, pp. 814 to 818.

The Three Cardinal Forms of X-Ray Findings in Silicosis (Rept. 1): Harunori Asakawa, pp. 819 to 828.

On the Radioactive Contamination and Removal of It (Rept. 1): *The Contamination of Wood with CA^{45} and Removal of It.* Norio Isida, pp. 836 to 845.

V. THE JOURNAL OF SCIENCE OF LABOUR (JAPAN). Vol. 32, No. 1 (1956).

On the Air Flow Resistance of Respiratory Protective Equipment. Studies on the Dust Respirators (Rept. 3). Toyohiko Miura, Mituo Morioka and Kikuzi Kimura, pp. 1 to 10.

On the Tuberculosis Changes of the Lungs as Complications of Silicosis. Takashi Hirokawa, pp. 11 to 42.

The Three Cardinal Forms of X-Ray Findings in Silicosis (Rept. 2). H. Asakawa, pp. 43 to 51.

Three Cases of Methyl-Bromide Poisoning. Hiroshi Takasaki, Minoru Sugimoto and Seizo Ikuta, pp. 70 to 76.

The authors found BAL was remarkably effective as therapy.

VI. CHRONICLE OF THE WORLD HEALTH ORGANIZATION. Vol. 10, No. 1 (1956).

Occupational Health. pp. 14 to 20.

This is a brief summary of a 251-page report from the *Bull. Wld. Hlth. Org.*, Vol. 13, 1955. The original report contains a

selection of papers presented at the Leyden seminar, December, 1952, and the Milan seminar, October, 1953, on the development of occupational health services in all countries.

A high rate of industrial accidents is found in many countries. For example, in France alone, among six million workers covered by social security, some 1,800,000 persons annually suffer occupational accidents, of whom 3,000 die, 70,000 are permanently disabled and 100,000 are temporarily disabled.

Factors are discussed such as organizing work with due regard to human physiology and psychology, the difficulty to enforce safety rules, and the doubtful value (or even bad effect) of posters to prevent accidents. It is stated that the higher the profession category in one country, the less is the attention given to safety measures and accident prevention.

◆ News of Local Sections

Michigan (Detroit)

THE SPEAKER at the meeting March 20, was LUCIEN BROUHA, M.D., Physiologist, Haskell Laboratory for Toxicology and Industrial Medicine, E. I. du Pont de Nemours & Company. The subject of his talk was "The Effects of Heat on Workers." He discussed such things as evaluation of heat problems, the criteria for gauging the extent of the problem, and the methods for controlling adverse effects of heat on workers.

New Officers for the 1956-1957 year were inaugurated at the meeting on March 20. They are: President, Fred T. McDermott, Michigan State Health Department; President-Elect, PAUL E. TOTH, Chrysler Corporation; Secretary-Treasurer, VINCENT J. CASTROP, General Motors Corporation.

Western New York

DR. HAROLD C. HODGE and DR. JAMES H. STERNER were speakers at the meeting on March 7, held at the University of Rochester School of Medicine and Dentistry. Their subject was "The Development and Use of Threshold Limits." DR. HODGE spoke on contributions from experimental toxicology and DR. STERNER on contributions from environmental and clinical studies.

New officers installed on March 7, for the year 1956-1957 are as follows: President, ROBERT H. WILSON; President-Elect, WILFRED L. MCMAHON; Secretary-Treasurer, FRANKLIN A. MILLER; Directors, SANFORD I. HARTWELL, EDWARD C. RILEY and DR. F. J. WEISMILLER.

St. Louis

PAUL HALLEY, Industrial Hygienist, Standard Oil Company of Indiana, was the speaker at the March 27 meeting. MR. HALLEY discussed industrial hygiene problems associated with the petroleum industry. A lively discussion followed his interesting presentation.

Officers for 1956-1957 are: Chairman, C. H. MEHAFFEY; Chairman-Elect, JACK T. GARRETT; Director and Executive Committee Member, FRED M. YOUNGER; Secretary and Treasurer, MRS. ALICE C. DEVERS, R.N.

Gulf Coast

AN INFORMAL Discussion Panel on Plant Health Problems was held April 10, at the L & C Cafeteria, Houston. Management, medicine, nursing, safety engineering and industrial hygiene were represented on the panel which conducted a most interesting and informative discussion. Panel members were: CARL A. NAU, M.D., Professor of Public Health and Industrial Medicine, University of Texas Medical School, Galveston, Texas; JALANNE GARRETT, R.N., Industrial Nurse, Westinghouse Electric Corporation Houston; RALPH JOHNSON, Supervising Engineer, Liberty Mutual Insurance Company, Houston; C. M. FERGUSON, President, Federal Steel Products Corporation, Houston; HARVEY R. MOBLEY, Industrial Hygienist, City Health Department, Houston.

Southern California

JOHN T. MCLAUGHLAN, M.D., spoke on "The Eye Protection Program at Hughes Aircraft Corporation" at the meeting held May 10, at Botwin's Restaurant, Los Angeles. DR. MCLAUGHLAN discussed the cost of the medical program at Hughes and presented his own motion picture film on eye protection.

St. Louis

A PANEL on Industrial Noise presented an interesting program at the meeting May 22, held at the Melbourne Hotel. Panelists were HAZEL LESLIE, R.N., U.S. Defense Corporation;

J. W. WEST, M.D., Otologist, St. Louis; J. R. COX, JR., Ph.D., Research Physicist, Central Institute for the Deaf; STEPHAN MILLET, Chairman, Industrial Commission of Missouri.

Northern California

PROFESSOR D. M. FINCH, Professor of Electrical Engineering, University of California, was the speaker at the meeting held March 13, at the Ed Jardin Restaurant, San Francisco. His subject was "Noise Evaluation in Industry." His talk centered about the technical aspects of noise such as the units used for measurement; why these units are used; the significance of the decibel, phon, sone and frequency. Slides were used to bring forth salient points in a very descriptive manner.

At the March 13 meeting, six were approved for Associate membership—three physicians, two engineers and one chemist.

A luncheon meeting was held April 13, at Spenger's Fish Grotto, Berkeley, at which time WELDON L. RICHARDS discussed the "Bay Area Air Pollution Control District," of which he is a board member. He told of the formation of the district, the objectives and the progress being made. A stimulating question period followed the talk.

At Spenger's Fish Grotto on the May 8 meeting, the speaker was J. E. GILMORE, Safety Director, Johns-Manville Lompoc Plant. He gave an interesting discussion of the elaborate engineering controls devised and used for the control of diatamaceous earth dust exposures during mining and processing. The speaker stated that the ¾ square mile area at Lompoc is the largest and purest diatomite deposit in the country. The company has established 107 sampline stations at which they obtain more than 800 dust count samples annually. Large sums have been spent for dust control.

Chicago

ELMER P. WHEELER, Assistant Medical Director, Monsanto Chemical Company, St. Louis, spoke on "Health and Safety Problems in the Production of a New Plastic—Urethanes" at the meeting held March 7. MR. WHEELER'S discussion was supplemented with a movie showing manufacturing operations. Several samples of the new plastic were displayed which indicate that this material has wide versatility.

Election of officers was held March 7, at which time F. A. VAN ATTA, Research Director, National Safety Council, became President-Elect, and Al Edwards, Industrial Hygienist, Portland Cement Association, became Secretary-Treasurer. Elected to the Board of Directors were MRS. VIRGINIA GALT, R.N., Bell & Howell Company; MELVIN RAMEY, International Harvester Company; and ROGER WILLIAMS, Chicago Eye Shield Company.

No local meeting was held in April due to the annual AIHA Convention. The last meeting until fall was held May 2, in Milwaukee through arrangements by the large membership in the Milwaukee area. The meeting was held at Steinhall of the Miller Brewing Company. The scientific part of the meeting was limited to a short talk by J. L. CANTRELL, Safety Director, Miller Brewing Company.

Industrial Hygiene Foundation

THE INDUSTRIAL HYGIENE FOUNDATION has completed 20 years of service to industry in the field of occupational health problems. A bulletin entitled "History of Industrial Hygiene Foundation" has been recently published by the Foundation. It describes the services and activities of the past 20 years. This bulletin is available at no charge. Requests should be addressed to the Industrial Hygiene Foundation, Mellon Institute, 4400 Fifth Avenue, Pittsburgh 13, Pennsylvania.

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